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2014 年度台灣再生醫學學會學術研討會暨會員大會
2014 Annual Meeting of Formosa Association of Regenerative Medicine
Scientific Program

Time	Topic	Speakers & Authors	Moderator
08:30 Registration 報 到			
Open Remark			
09:00~09:30 論文比賽 Oral Presentation Competition (I)			
S-1 09:00~09:10	Preparation and Investigation of Hyaluronic Acid-Based Hydrogel Containing Gelatin Particles for Cartilage Tissue Engineering	<u>鍾易龍</u> 方旭偉 林峰輝	嚴孟祿 教授 林泰元 教授
S-2 09:10~09:20	3D Printing from Green and Sustainable Nanoelastomers for Tissue Engineering Applications	<u>洪堃哲</u> 曾清秀 徐善慧*	
S-3 09:20~09:30	Visualization of Migration for Stem Cells Implanted in a Peripheral Nerve Conduit Using MRI	<u>曾庭箴</u> 徐善慧*	
09:30~12:10 Invited Lectures			
I-1 09:30~09:55	Mapping Melanocyte Stem Cell Activities During Feather Pigment Pattern Formation	林頌然	侯連團 教授
I-2 09:55~10:20	Endothelial Progenitor Cell and Cardiovascular Disease – A Marker for Vessel Health	黃柏勳	
10:20~10:40 Coffee Break			
I-3 10:40~11:05	Human Experienced Medicine Based Regenerative Drug Discovery: Anti-Aging Herbs to Panacea, Fact or Fiction ?	吳榮燦	林敬哲 教授 何美冷 教授
I-4 11:05~11:30	Induction of Cells Into Senescence by PTTG1 and its Implication in Tumorigenesis	林敬哲	
I-5 11:30~12:10	Telomerase Activation in Telomere Replication	鄧述諄	
12:10 會 員 大 會			
12:10~13:30 Lunch Break			

Time	Topic	Speakers & Authors	Moderator
13:30~15:00 Invited Lectures			
I-6 13:30~14:10	Visible Light in Regenerative Medicine	余幸司	劉華昌 教授 陳敏慧 教授
I-7 14:10~14:35	青春的泉源--女性荷爾蒙	嚴孟祿	
I-8 14:35~15:00	促皮膚修復化粧品之現狀簡介	邱品齊	
15:00~15:30 Coffee Break			
O-1 15:30~15:45	Molecular Weight of Hyaluronan Alters its Potential of Chondrogenic Effect on Adipose Derived Stem Cells	吳順成 林怡珊 張智翔 張瑞根 王國照 何美玲	徐善慧 教授
O-2 15:45~16:00	Using Non-proteinous Drugs as Bio-factors to Enhance Chondrogenesis of hADSCs in HA-enriched Microenvironment for Tissue Engineering	張智翔 吳順成 張瑞根 陳崇桓 何美玲	
16:00~16:40 論文比賽 Oral Presentation Competition (II)			
S-4 16:00~16:10	Enhancement of Renal Epithelial Cell Functions through Microfluidic-based Coculture with Adipose-derived Stem Cells	黃惠君 張雅茹 陳宛君 韓逸成 湯銘哲 吳佳慶	嚴孟祿 教授 林泰元 教授
S-5 16:10~16:20	Cultivated Dermal Fibroblast Spheroids for Corneal Stromal Tissue Reconstruction	王沛然 賴瑞陽*	
S-6 16:20~16:30	Influence of Matrix Nanostructure on the Functionality of Carbodiimide Cross-Linked Amniotic Membranes as Limbal Epithelial Cell Scaffolds	羅麗娟 賴瑞陽*	
S-7 16:30~16:40	Functional Recoveries of Sciatic Nerve Regeneration by Combining Chitosan-Coated Conduit and Neurosphere Cells Induced From Adipose-Derived Stem Cells	薛元毓 張雅茹 黃子捷 范詩辰 王鐸翔 陳家進 吳佳慶 林聖哲	
論文比賽頒獎			
Close Remark			

Invited Lectures

09:30-09:55

I-1

Mapping Melanocyte Stem Cell Activities During Feather Pigment Pattern Formation

Sung-Jan Lin, MD. PhD.

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National Taiwan University, Taipei, Taiwan

The diversity of feather pigment patterns has amazed many, but the identity of feather melanocyte stem cell (McSC) and the mechanisms regulating pigment patterns have been unveiled little. We show that McSCs are arranged as a ring around the proximal collar bulge epithelium, continuously sending out progeny distally to paint the differentiating keratinocytes in growing stage. In resting stage, this circular niche descends to the lowest tips of papilla ectoderm and McSCs become quiescent. The unique cylindrical plane formed by McSCs and their progeny yields new dimensions of regulatory possibilities that are constrained by the highly localized McSC niche topology in mammalian hair follicles. For pigment switch, all whites observed in various pigment patterns are not equal. They are created by several basic cellular mechanisms including McSC removal, suppressed melanocyte emigration or inhibited differentiation. Variation in temporal and spatial employment of these cellular mechanisms helps to create pigment patterns. We also found an unexpected role of feather mesenchymal pulp cells in regulating pigment patterns by inhibiting melanocyte differentiation through patterned expression of agouti signaling protein. Feather pulp cells are also able to respond to physiological changes, such as sexual maturity, to change pigment patterns in regenerating feathers through varying temporal and spatial agouti expression patterns. Hence, the complex feather pigment patterning on the cylindrical epithelial canvas is achieved by multiple-dimensional co-option of basic cellular mechanisms during evolution.

09:55-10:20

I-2

**Endothelial Progenitor Cell and Cardiovascular Disease –
A Marker for Vessel Health**

Po-Hsun Huang, MD, PhD
Associate Professor

Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan;
Division of Cardiology, Taipei Veterans General Hospital, Taipei, Taiwan.

Atherosclerosis is a systemic inflammatory disease of arterial wall and initiated by endothelial damage. The integrity and functional activity of endothelial monolayer play an important role in atherogenesis. The extent of endothelial injury may represent a balance between the magnitude of injury and the capacity for repair. Traditional view suggested endothelium integrity is maintained by neighboring mature endothelial cells which migrate and proliferate to restore the injured endothelial cells. However, a series of clinical and basic studies prompted by the discovery of bone marrow-derived endothelial progenitor cells (EPCs) have demonstrated that the injured endothelial monolayer may be regenerated partly by circulating EPCs. These circulating EPCs are mobilized endogenously triggered by tissue ischemia or exogenously by cytokine stimulation. Clinical studies demonstrated that levels of circulating EPCs are associated with vascular endothelial function and cardiovascular risk factors, and help to identify patients at increased cardiovascular risk. Reduced levels of circulating EPCs independently predict atherosclerotic disease progression and development of cardiovascular events. There are several ways to increase levels of circulating EPCs and improve their function by pharmacological strategies and lifestyle modification. Enhancement of the regenerative capacity of the injured endothelium seems one way to reduce the incidence of atherosclerotic lesions. Therefore, a better understanding of the relation between EPCs and atherosclerosis would provide additional insight into the pathogenesis of cardiovascular disease and create novel therapeutic strategies.

10:40-11:05

I-3

以人体使用經驗進行再生醫學新藥開發:由抗老化中藥到萬靈藥的真實與夢幻?
**Human Experienced Medicine Based Regenerative Drug Discovery:
Anti-aging Herbs to Panacea, Fact or Fiction ?**

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Rong-Tsun Wu, Research Center for Drug Discovery,
Institute of Biopharmaceutical Sciences, National Yang-Ming University

There are many traditional and anecdotal claims to the therapeutic properties of ethnobotanical drugs. The prevalence of age-related pathologies, such as cardiovascular disease, neurodegenerative disease and diabetes type II, has increased dramatically with the rising average age of populations. Regenerative medicine aims to develop new therapies through the stimulation of natural regenerative processes in human beings. Our body endogenously produce many factors gives rise to autocrine and/or paracrine signaling in different organs particularly during hypoxia, inflammatory and injury conditions for tissue or organ regeneration. Here, we address the function of nerve growth factor (NGF) in neuronal cells and erythropoietin (EPO) in non-hematopoietic cells as the regenerative factors for age-related diseases, and highlight the endogenous NGF or EPO inducers from human experienced medicine will provide important clues to develop drugs for age-related diseases.

We found that NGF enhanced mitochondrial biogenesis through activating PGC-1 α in PC12 neuronal and astrocytes cells, and NGF treatment could reduce Huntington aggregates through autophagy induction in a cellular Huntington model. We furtherly found a famous anti-aging Chinese herb *Ganoderma lucidum* (GaLu) as an astrocytic NGF inducer, both the extract and its active constituents had potent effects. After the mitochondrial toxin 3-nitropropionic acid (3-NP) induced behavioural impairment and striatal degeneration, GaLu treatment therapeutically restored the behaviour score, sensorimotor ability and neuronal loss in this HD mouse model. We also found that striatal NGF, PGC-1 α mRNA expression and succinate dehydrogenase activity were recovered in the GaLu-fed HD mice. Our data suggest that the modulation of endogenous NGF signalling may offer an effective approach for neuroregeneration in mitochondrial-related diseases, such as Huntington's disease, ischemia, Alzheimer's disease and Parkinson's disease.

We also demonstrated that a small molecule inducer of EPO isolated from *P. multiflorum* Thunb, EH-201, activated EPO-mediated mitochondrial function and hemoglobin expression in C2C12 myocytes, kidney slices, and hepatocytes. EH-201 robustly enhanced the endurance performance activity during hypoxia stress in both healthy and doxorubicin-induced cardiomyopathic mice with increasing myocardial hemoglobin expression and mitochondrial biogenesis, and resulting improvement of cardiac damages. EH-201 also ameliorated the anemia and renal failure in cisplatin-induced nephropathic mice. Thus, the enhancement and recovery of cellular

oxygenation through stimulating mitochondrial activity and hemoglobin production in non-hematopoietic cells by an endogenous EPO inducer might serve as a potential therapeutic strategy for age-related diseases as dry eye, chronic heart failure, aging related neurodegenerative diseases, age-related macular degeneration, chronic obstructive pulmonary disease, chronic kidney disease, and anemic cancer patients undergoing chemotherapy. We shed light the active ingredient EH-201 from anti-aging herb to panacea for age-related diseases is a fact, not fiction.

11:05-11:30

I-4

Induction of Cells Into Senescence by PTTG1 and its Implication in Tumorigenesis

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Abstract

Under normal culture conditions, normal diploid fibroblasts undergo a limited number of cell divisions and then cease proliferation. It is well documented that cellular senescence is caused by attrition of telomeres after repeated cycles of cell divisions. The mechanism of how senescence is induced is less clear. I will discuss several of our attempts to understand the mechanism of senescence. Among these attempts, I will focus on pituitary tumor transforming gene (*PTTG1*, securin) to discuss its role in senescence. *PTTG1* is involved in cell-cycle control through inhibition of sister-chromatid separation. Elevated levels of *PTTG1* were found to be associated with many different tumor types that might be involved in late stage tumor progression. We found expressing *PTTG1* in normal human fibroblasts inhibited cell proliferation. Several senescence-associated (SA) phenotypes including increased SA- β -galactosidase activities, decreased BrdU incorporations, and increased SA-heterochromatin foci formation were also observed in *PTTG1*-expressing cells, indicating that *PTTG1* overexpression induced a senescent phenotype in normal cells. Significantly, the *PTTG1*-induced senescence is p53-dependent and telomerase-independent, which is distinctively different from that of replicative senescence. We also provide evidence that *PTTG1*-overexpression caused chromosome instability, which subsequently induced p53-dependent senescence through activation of DNA-damage response pathway. The role of *PTTG1* in initiating tumorigenesis will also be discussed.

11:30-12:10

I-5

Telomerase Activation in Telomere Replication

Shu-Chun Teng

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Telomeres are dynamic DNA-protein complexes that protect the ends of linear chromosomes. Most telomeric DNA is synthesized by the enzyme telomerase. While most somatic cells do not express telomerase and therefore have limited life span, cancer cells can bypass the crisis mainly through telomerase reactivation.

The Krüppel-like transcription factor 4 (KLF4) has been implicated in cancer formation and stem cell regulation. However, the function of KLF4 in tumorigenesis and stem cell regulation are poorly understood due to limited knowledge of its targets in these cells. We have revealed a surprising link between KLF4 and regulation of telomerase, which offers important insight into how KLF4 contributes to cancer formation and stem cell regulation. KLF4 directly activate expression of the human telomerase catalytic subunit, hTERT. Our findings demonstrate that *hTERT* is one of the major targets of KLF4 in cancer and stem cells to maintain long-term proliferation potential.

Moreover, we demonstrated that telomerase is mainly activated by Cdk1/Tel1/Mec1 on telomeric binding protein Cdc13 from late S to G2 phase of the cell cycle. Cdk1 phosphorylates residues 308 and 336 of Cdc13. The residue T308 of Cdc13 is critical for efficient Mec1-mediated S306 phosphorylation. Phenotypic analysis *in vivo* revealed that the mutations in the Cdc13 S/TP motifs phosphorylated by Cdk1/Mec1/Tel1 caused cell cycle delay and telomere shortening and these phenotypes could be partially restored by the replacement with a negative charge residue. Furthermore, the Cdk1-mediated phosphorylation was required to promote the regular turnover of Cdc13. Hypernegatively charged domain of Cdc13 contributed by Cdk1, Tel1 and Mec1 may provide an optimal interface to recruit the potential positively charged domain near the amino acid 444 lysine residue of Est1 in the telomerase complex. Together these results demonstrate that Cdk1/Mec1/Tel1 phosphorylate the telomerase recruitment domain of Cdc13, thereby preserves optimal function and expression level of Cdc13 for precise telomere replication and cell cycle progression.

13:30-14:10

I-6

Visible Light in Regenerative Medicine

Hsin-Su Yu, M.D., Ph.D.

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Phototherapy plays important therapeutic role in different medical fields including regenerative medicine. In 2003, we proposed and demonstrated that low-energy Helium-Neon (He-Ne) laser emitting light at 632.8 nm (red light) induces repigmentation of vitiligo in clinical practice. Mechanistically, we elucidated that He-Ne laser initiates mitochondrial retrograde signaling via a Ca^{2+} -dependent cascade, leading to activation of peroxisome proliferators-activated receptor γ coactivator-1 α (PGC-1 α), the master regulator for mitochondrial biogenesis and antioxidant enzyme production, and ultimately resulting in melanoblast cell differentiation. These studies provide clinical and scientific evidences indicating that visible light is useful in the field of regenerative medicine.

Another clinical application of visible light is to reduce sun-associated aging by decreasing metalloproteinase-1 (MMP-1) expression in the dermis. It is known that UVA-associated skin aging is mediated by increased MMP-1 expression via reactive oxygen species formation. Very recently, we demonstrated that pretreating dermal fibroblasts with visible light at 590 nm attenuated the UVA-induced expressions of ROS and MMP-1. The increased expression of PGC-1 α again played a crucial role in this process by upregulating the expression of antioxidant enzymes.

In summary, visible light phototherapy have important clinical applications in the regenerative medicine since visible light enhances mitochondrial biogenesis and reduces oxidative stress via regulating mitochondria-associated signaling events. With clarification of mechanisms involved, it is foreseeable that visible light therapy will gain more importance in the field of regenerative medicine in the near future.

14:10-14:35

I-7

青春的泉源 -- 女性荷爾蒙

嚴孟祿醫師
台大醫院婦產部

老化是一種自然的過程，長生不老至今仍是神話，只要是就會老，只是速度有快有慢。老化雖然是自然的過程，但卻不是個令人愉快的狀態。

女性因為停經後，女性荷爾蒙會突然快速減少，故常會因為缺乏女性荷爾蒙，出現更年期症狀及骨質疏鬆症。男性的荷爾蒙則是緩慢逐步的降低，不像女性會突然的減少，所以比較沒有明顯的更年期症狀。

女性在雙十年華時皮膚柔潤有彈性，老年時變得雞皮鶴髮，究其原因主要就是在於「女性荷爾蒙」的有無。女性荷爾蒙可以維持水分及膠原蛋白在皮膚中，使皮膚顯得光滑有彈性。現代科學也證實，女性荷爾蒙的缺乏是造成女性衰老的重要原因，事實上我們現在已經知道女性荷爾蒙是女性賴以發育和保持青春特徵的最重要物質。除此之外它還具有許多生理功能：能夠增加體內有益的膽固醇濃度並降低不好的膽固醇濃度，因此能夠減少血管硬化的機會；能減少骨質流失的速率；保持陰道潤滑俾能享受性生活；防止乳房的鬆弛下垂及減緩皮膚的老化過程等。

目前的實證醫學知道：婦女要減緩更年期的不適需要補充女性荷爾蒙，如果子宮還在的，還要加上黃體素以保護子宮內膜，但這樣使用超過三年會增加乳癌的危險。但是如果子宮已切除者，則只需要補充女性荷爾蒙即可，有趣的是這樣反而會略為減少罹患乳癌的危險，因此子宮已切除者較傾向於可以補充女性荷爾蒙。至於仍保有子宮的更年期婦女，如果沒有明顯更年期的症狀，因有上述的疑慮，目前醫學上並不建議使用荷爾蒙補充療法。但對於有明顯更年期症狀者，荷爾蒙補充療法恐怕是目前醫學上唯一被證實有效且建議使用的方法。只是為降低罹患乳癌的危險，應該慢慢地將劑量調整到沒有更年期症狀的最低劑量，且儘量在三年內結束用藥，這樣才能得到荷爾蒙補充療法的好處而儘量避免它的壞處。

想留住青春是很多人的期望。因此坊間充斥著許多抗老療法。抗老療法可大致分成兩大類，一類是外科美容整形，包括拉皮、除斑、注射膠原蛋白等，另一類就是藉由服用或注射各種藥物，企圖改變生理狀況。因為女性荷爾蒙確實具有保持青春的效果因此常被使用，但因它有增加乳癌的副作用，故需在醫師謹慎評估下小心使用。至於另外有些偏方，譬如有人注射「生長素」來抗老，使脂肪減少讓體態健美，可是這仍有誘發腫瘤的疑慮。另外注射「胎盤素」更是流傳已久的老偏方，但因它是注射外來的異物，要小心有嚴重過敏的可能，故這些方法都尚未通過安全確認，目前較有實證醫學根據且能為我們掌控的青春泉源，仍非女性荷爾蒙莫屬！

14:35-15:00

I-8

促皮膚修復化粧品之現狀簡介

邱品齊 醫師
台大醫院雲林分院皮膚部

摘要:

近年來皮膚再生醫學已經成為皮膚科學顯學，各式各樣促進皮膚修復的醫療處理也陸續在臨床上應用，無論是雷射、敷料、高壓氧以及藥品都有其特殊角色。

然而化粧品近年來的進步也是不可同日而語，相關生技與植萃成分的應用更是日新月異，功能迥異的活性成分也常被市場所應用。雖然化粧品在法律上不能涉及療效，但由於藥妝品(功能性化粧品)概念的興起，也讓化粧品多了很多特殊的用途。

本演講將會說明目前市場上促皮膚修復化粧品的應用，以及使用在其中的相關成分。此外這類化粧品常見誇大不實的廣告案例也會一併說明，希望可以讓大家對這議題有客觀的認識與了解。

Oral Presentations

15:30-15:45

O-1

玻尿酸的分子量可改變對於脂肪幹細胞軟骨化的能力
**Molecular Weight of Hyaluronan Alters its Potential of Chondrogenic Effect on
Adipose Derived Stem Cells**

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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
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Introduction: Guiding stem cells differentiate into chondrogenic lineage is important for adipose derived stem cells based articular cartilage tissue engineering. Appropriate cell-niche interaction may be used for promoting chondrogenic differentiation of stem cells. Our previous finding indicates that hyaluronan(HA)-microenvironment initiates chondrogenesis of adipose derived stem cells (ADSCs). However, different molecular weight (M.W.) of HA on the chondrogenic differentiation of ADSCs is still undefined. This study was to search the appropriate molecular weight of HA for an optimal effect on initiating and enhancing chondrogenesis in ADSCs.

Materials and Methods: ADSCs were isolated from the adipose tissue of New Zealand white rabbit. Osteogenesis, chondrogenesis, and adipogenesis were induced to characterize the multilineage potential of the ADSCs. To investigate the effect of chondrogenic potentials of different M.W. of HAs, the solutions of HA (The M.W. of HA are 8000, 0.6 million, and 2 million) were coated on culture well before cell seeded on wells. Twenty four hour after ADSCs seeded on HA-coated well, cell aggregation of ADSCs were observed. The cartilage nodule formation and Sulfated glycosaminoglycan (sGAG) synthesis of ADSCs was determined by Alcian blue staining and guanidine-HCl extraction method 5days after cell seeded. For in vivo study, ADSCs mixed with HA (M.W. : 2 million) were encapsulated in fibrin hydrogel (ADSC/HA) and then implanted into the articular cavity of knee of rabbits.

Results: The ADSCs can be induced into osteogenesis, chondrogenesis and adipogenesis, indicating the ADSCs possess multilineage potential. 24 hours after ADSCs seeded on HA-coated wells, cell aggregations were induced by HA with molecular weights of 8000, 0.6 million and 2 million Da. The 2 million Da HA treated group showed optimal effect on cell aggregation. More pronounced cartilage nodule formations and sGAG depositions were also found in 2 million Da HA treated groups. The in vivo study showed that the construct of ADSC/HA of 2 million M.W. promoted neo-cartilage formation in joint cavity.

Discussion: Two million M.W. of HA showed the optimal effect on promoting chondrogenesis. In vivo study also showed that within the articular cavity environment, the ADSCs encapsulated in HA-enriched fibrin gel were capable of forming neo-cartilage in vivo.

Conclusions: Based on these findings, we suggest that 2 million M.W. of HA provides better cell-niche interaction that may be an alternative choice for directing chondrogenic differentiation of ADSCs in ADSC based articular cartilage tissue engineering.

15:45-16:00

O-2

以非蛋白質藥物作為生物因子於玻尿酸微環境中促進幹細胞軟骨分化之組織工程研究
Using non-proteinous Drugs As Bio-factors to Enhance Chondrogenesis of hADSCs in
HA-enriched Microenvironment for Tissue Engineering

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高雄醫學大學醫學系生理學科⁴

Introduction : The aim of this study is to search a combination use of hyaluronic acid and small-molecule drugs to enhance chondrogenic differentiation of hADSCs for articular cartilage tissue engineering. In this project, we found the optimal effective timing and concentration of Alendronate (Aln) and Simvastatin (Sim) for enhancing hADSCs chondrogenesis both in 2-D and 3-D culture *in vitro*. The ongoing work is to test the chondrogenic effect of Sim, carried by a short-term controlled release drug carrier, in combination with HA-based hydrogel on human ADSCs *in vivo*.

Materials and Methods : Human adipose derived stem cells (hADSCs) were isolated from patients' adipose tissue undergoing surgery with informed consent. Isolated hADSCs were cultured in HA-coated or non-coated plates treated with various doses of drug A or B. In three-dimensional culture, hADSCs were seeded in HA/Fibrin hydrogel and treated with drug B. The fresh medium was changed every two days. The chondrogenic genes expression were measured by q-PCR, including Sox-9, Aggrecan and Col II. The sulfated glucosaminoglycan (sGAG) formation was examined by dimethylmethylene blue (DMMB) assay and alcian blue staining. The type II collagen expression was measured by ELISA. Statistical analyses were performed using Student's t -test, with p values below 0.05 being considered significant.

Results : In this project, we have investigated: 1) The chondro-differentiation effects of Aln and Sim were investigated under the HA-enriched micro-environment. The cell aggregation was pronounced in drugs treated, HA-coated group. The extracellular matrix, including sGAG and type II collagen were elevated in Sim-treated groups. 2) The chondrogenic genes expression, including Sox-9, type II collagen and aggrecan were enhanced in Aln or Sim treated hADSCs in HA-enriched micro-environment. 3) Sim was encapsulated in PLGA microsphere and measured the releasing profile for two weeks. The SEM pictures showed the slow-degradation of microspheres and the HPLC analysis showed the drug was released with time in the effective dose range. 4) The matrix formation was measured by DMMB assay and showed the HA/Fibrin/hADSCs/Sim-carrier groups presented higher sGAG level than control group.

Discussion : Construction of a 3D biomaterial with autologous adult stem cells to regenerate defected articular cartilage may become a viable clinical option. In this study, we first examined HA can be a enhancing factor for ADSCs to differentiate during chondrogenesis by using HA-coated wells or hydrogel, and demonstrated that the Sim supplied in a HA-enriched microenvironment further enhanced cell chondrogenic differentiation.

Conclusions : In this proposed project we demonstrated the effect of statin and bisphosphonate for ADSCs chondrogenesis *in vitro* in a 2-D and 3-D culture with HA-based hydrogel. Our results emphasize that these non-protein drugs enhanced hADSCs chondrogenesis capacity a HA-enriched microenvironment. It may be applied for more effective articular cartilage tissue engineering.

S-1

09:00-09:10

透明質酸水膠結合明膠顆粒應用於軟骨組織工程之製備及性質研究
**Preparation and Investigation of Hyaluronic Acid-Based Hydrogel Containing Gelatin
Particles for Cartilage Tissue Engineering**

鍾易龍¹ 方旭偉² 林峰輝³

國立台北科技大學生化與生醫工程所¹ 國立台北科技大學化學工程與生物科技系²
國立臺灣大學醫學工程研究所³

Introduction : In recent years, the cartilage injury repair has been a popular topic in regenerative medicine. The cartilage has limited self-repair capacity due to lack of nerves and blood vessels in the articular cartilage. So far, some treatment surgery have been developed in clinic; however, some limitations still exist such as fibrocartilage formation. Until the tissue engineering is well developed, cartilage repair will reach to a higher level. To date, the most suitable bioscaffold for chondrocytes in the cartilage repair is still not finding. In this study, we have developed the hyaluronic acid-based hydrogel and incorporated it with gelatin particles to evaluate the possibility as the bioscaffold for cartilage tissue engineering. We expected the bioscaffold not only mimic ECM functions, but also have the ability to promote chondrocytes proliferation and maintain the phenotype.

Materials and Methods : In this study, the oxidized hyaluronic acid with aldehyde functional groups prepared by sodium periodate was crosslinked by adipic acid dihydrazide (ADH) to form hydrogel. The gelatin was modified by glutaraldehyde and proanthocyanidins crosslink agents to form gelatin particles. Then we incorporated hydrogel and particles as the bioscaffold. To investigate the characterization of bioscaffold, the IR、SEM、EDS、degradation time and biocompatibility test were evaluated.

Results : The results shown the gelatin particles had uniform pore size about 80-150 μm and the crosslinking index remained above 90%.The hydrogel containing gelatin particles through WST-1, LDH and Live&Dead analysis are non-cytotoxicity ; also has good biocompatibility.

Discussion and Conclusions : In this study, we prepared hyaluronic acid-based hydrogel containing gelatin particles as chondrocytes bioscaffold. The oxidized hyaluronic acid /ADH crosslinked hydrogel can be prepared in a liquid form at room temperature and easily mixed with gelatin particles and chondrocytes. These preliminary data indicate that the hydrogel and gelatin particles have good biocompatibility, and may have potential uses in cartilage tissue engineering applications.

09:10-09:20

S-2

三維列印之生物可分解奈米彈性體於組織工程之應用
3D Printing from Green and Sustainable Nanoelastomers for Tissue Engineering Applications

洪堃哲¹ 曾清秀² 徐善慧^{1*}
國立臺灣大學高分子科學與工程學研究所¹
國立中央大學機械工程學系²

Introduction : Three-dimensional (3D) printing is a rapid prototyping and fabrication technology. Scaffolds can be fabricated by 3D printing method. However, the toxic photo-initiator, heat or toxic organic solvents would be used. Biodegradable polymers such as polylactide and polyglycolide have been used to make scaffolds. Nevertheless, these materials are in lack of the proper elasticity that matches to that of living tissue. In this study, a novel biodegradable waterborne PU was synthesized in the form of nanoparticles (NPs). We then employed a 3D printing strategy to fabricate elastic scaffolds from the water dispersion of PU. We attempted to establish a green and sustainable 3D printing platform to derive non-toxic and highly elastic scaffolds for customized tissue engineering applications.

Materials and Methods : The biodegradable PU elastomers were synthesized from a water-based process. The soft segment was two oligodiols, poly(ϵ -polycaprolactone) diol and polyethylene butylene adipate diol. The hard segment was isophorone diisocyanate, 2,2-bis(hydroxymethyl) propionic acid and ethylenediamine. 3D scaffolds were printed from a feed containing PU NPs dispersion and polyethylene oxide (PEO). The rheologic properties of PU/PEO solutions were determined by rheometer. The mechanical properties and degradation rate of scaffold were evaluated. The scaffolds were seeded with chondrocytes for evaluation of their potential as cartilage scaffold.

Results : A 3D scaffold was successfully fabricated by 3D printing process from the dispersion of PU NPs. The elastic recovery of PU scaffolds was significantly better than that of polylactic-co-glycolic acid (PLGA) scaffolds made from the solution in organic solvent. The compressive modulus was close to that of native cartilage. The seeding efficiency, proliferation, and matrix production of chondrocytes in PU scaffolds after 7 and 14 days were superior to those in PLGA scaffolds.

Discussion : Elastic biodegradable scaffolds were fabricated by 3D printing. Not any toxic organic solvent, crosslinker, or initiator was used. The environment-friendly process generated a highly elastic, force-transducing scaffold with good affinity to cells.

Conclusions : The green and sustainable 3D printing platform offers a useful way to fabricate biodegradable/elastic scaffolds for tissue engineering applications.

09:20-09:30

S-3

利用核磁共振造影觀察幹細胞在周邊神經導管中的移動行為
**Visualization of Migration for Stem Cells Implanted in a Peripheral
Nerve Conduit Using MRI**

曾庭箴 徐善慧*

國立臺灣大學高分子科學與工程學研究所

Introduction : Magnetic resonance imaging (MRI) has been proven to be effective in tracking magnetic-labeled cells and evaluating their clinical relevance after cell transplantation. Our previous study has shown that nanoparticles (NPs) and genes can be delivered to mesenchymal stem cells (MSCs) during spheroid formation on chitosan-based substrates. The current study focused on tracking the Fe₃O₄ NP-labeled MSCs within a peripheral nerve conduit implanted in a rat sciatic nerve gap using MRI.

Materials and Methods : MSCs were labeled with Fe₃O₄ NPs and transfected with brain-derived neurotrophic factor (BDNF) gene by increased endocytosis during spheroid formation. Rat sciatic nerve was transected to create 10 mm gap, where a polylactide conduit was implanted to bridge the nerve. The Fe₃O₄ NPs-labeled MSC single cells or spheroids (with or without BDNF transfection) were injected in the conduit. The transplanted MSCs were tracked by MRI in vivo at 3, 10, 21, and 31 days. Prussian blue staining and histological analysis were performed after 31 days of implantation.

Results : Fe₃O₄ NP-labeled MSCs were successfully visualized by MRI, especially for both groups of MSC spheroids. Based on MR images, Fe₃O₄ NP-labeled MSC spheroids or BDNF-transfected MSC spheroids were more centrally located in the mid-portion of the conduit after 3 days. The dark signals in both of these groups then extended and spanned both ends at 10, 21, and 31 days. On the other hand, Fe₃O₄ NP-labeled MSC single cells were randomly spread in the conduit after 3 days and at 10 days, they were clearly observed at the proximal and distal portions of the injured nerve. No obvious dark signals were observed inside the conduit after 21 and 31 days. Animals receiving the conduits and BDNF-transfected spheroids had the shortest nerve connection time (< 21 days).

Discussion : Very early formation of fibrin cable (~ 10 days) and recruitment of MSCs along the cable were visualized for the sciatic nerve gap receiving MSC spheroids in a conduit. The gene modulation associated with neural development, neurotrophic factor, and chemokine receptor expressions may account for the observation. High-resolution MRI tracking of Fe₃O₄ NP-labeled cells offers useful information regarding how MSCs migrate during peripheral nerve regeneration.

Conclusions : The migration and distribution of the Fe₃O₄ NP-labeled MSCs in the injured peripheral nerve could be monitored by MRI. The substrate-mediated gene delivery and NP labeling may provide extra values for MSC spheroids to carry therapeutic/diagnostic agents in cell-based therapy

16:00-16:10

S-4

利用生物微機電技術研發工同培養之人工腎臟晶片
**Enhancement of Renal Epithelial Cell Functions through
Microfluidic-based Coculture with Adipose-derived Stem Cells**

黃惠君^{1,2} 張雅茹^{1,2} 陳宛君^{2,3} 韓逸成^{2,3} 湯銘哲^{2,3} 吳佳慶^{1,2,4,5}
國立成功大學細胞生物與解剖學研究所¹ 國立成功大學基礎醫學研究所²
國立成功大學生理學研究所³ 國立成功大學生物醫學工程學研究所⁴
國立成功大學前瞻醫療器材科技中心⁵

Introduction : Current hemodialysis has functional limitations and is insufficient for renal transplantation. The bioartificial tubule device has been developed to contribute to metabolic functions by implanting renal epithelial cells into hollow tubes and showed higher survival rate in acute kidney injury patients. The current study developed a microfluidic coculture platform to enhance epithelial cell function in bioartificial microenvironments with multiple microfluidics channels that are microfabricated by polydimethylsiloxane.

Materials and Methods : The adipose-derived stem cells (ASCs) were harvested from abdominal fat pad in S-D rats (8 weeks old). The coculture system was manufactured by BioMEMS technologies. The coculture platform was designed with the schematic of epithelial monolayer of Madin–Darby canine kidney (MDCK) cells to interact with ASCs embedded in 3D Collagen gel (CG). In comparison to coculture different cells using commercial transwell system, the current coculture device allowed living cell monitoring of both MDCK epithelial monolayer and CG-ASC in 3D microenvironment.

Results : By coculture with CG-ASC, the cell height was increased with columnar shapes in MDCK. Promotion of cilia formation and functional expression of ion transport protein in MDCK were also observed in the cocultured microfluidic device. When applying fluid flow, the intracellular protein dynamics can be monitored in current platform by using the time-lapse confocal microscopy and transfection of GFP-tubulin plasmid in MDCK.

Discussion : Since we only applied fluid flow rate at 0.025 mL/min in single microfluidic device and at least in the order of 10^5 flow rates should increase to provide sufficient fluid exchange if willing to connect current device with dialysis machine, the collection of fluid from multiple units of current microfluidic design is necessary for future hemodialysis. By distributive multiple branching of the microfluidic channels, the amplification of the coculture units to “array-like” chip could be achieved and allowed to inject/collect fluid simultaneously.

Conclusion : The schematic diagram of the current microfluidic device provides ASCs embedded in collagen gel and directly interacts with MDCKs. The cup-like peripheral channel allows the MDCKs to have a large interface with the central channel that contains CG-ASC. Coculturing with GC-ASC enhanced cilia formation and renal epithelial functions in MDCKs. The current coculture platform can apply shear stress on epithelial cells and allow fluid exchange. These advantages may provide beneficial innovations for developing renal chips in the future.

16:10-16:20

S-5

以皮膚纖維母細胞球體進行角膜基質組織重建

Cultivated Dermal Fibroblast Spheroids for Corneal Stromal Tissue Reconstruction

王沛然 賴瑞陽*

長庚大學生化與生醫工程研究所

Introduction: Currently, penetrating keratoplasty is the most common form of treatment for corneal dysfunction. However, the method involving allogeneic transplantation is limited by increasing shortage of donor cornea. In the study, we report the cultivation of dermal fibroblast spheroids for corneal stromal tissue reconstruction.

Materials and Methods: After plating of rabbit dermal fibroblasts (RDFs) on the hyaluronic acid (HA) coatings for 1-8 h, cell adhesion was carried out by scanning electron microscopy and immunocytochemistry. Then, the growth of multicellular spheroid aggregates was checked. To evaluate its extracellular matrix production capacity, the cultivated cell spheroid was studied by biochemical examinations. For in vivo experiments, the RDF spheroid aggregates were implanted in a rabbit model of bacterial keratitis. Postoperative outcomes were evaluated by means of clinical observations.

Results: The RDFs on HA coatings exhibited poor adhesion and tended to aggregate to form spheres. When compared to their counterparts in the form of dissociated cell suspensions, the RDF spheroids possessed better biosynthetic capacity. The injection of multicellular spheroid aggregates into the stromal defect could improve corneal clarity and function, indicating good tissue repair ability of these cell grafts.

Discussion: In clinical medicine, corneal stromal cells are not readily accessible, and it is difficult to obtain sufficient number of cells from patients. Therefore, an alternative strategy for corneal stromal reconstruction should be established to meet the increasing medical service needs. Here, we demonstrate the feasibility of cultivating dermal fibroblast spheroids on biomaterial coatings and transplanting these cell grafts to treat corneal wound.

Conclusions: Cultivated RDF spheroids may have potential for use in corneal stromal tissue reconstruction.

16:20-16:30

S-6

碳二亞胺交聯羊膜組織奈米結構對培養角膜輪部上皮細胞之影響評估
**Influence of Matrix Nanostructure on the Functionality of Carbodiimide
Cross-Linked Amniotic Membranes as Limbal Epithelial Cell Scaffolds**

羅麗娟 賴瑞陽*

長庚大學生化與生醫工程研究所

Introduction : Given that the collagen fibers constitute the architectural framework of amniotic membrane (AM), the nanoscale change in fibrous structure may induce substantial alteration of chemically modified biological tissues. It motivates us to study the relationship between nanostructure and functionality of carbodiimide cross-linked AM matrices as a limbal epithelial cell (LEC) scaffold biomaterials.

Materials and Methods : After cross-linking with carbodiimide for 1-4 h, the AM matrices were characterized by protein conformation, tissue ultrastructure, and substrate nanotopography. Samples with varying nanofibrous structures were evaluated by determinations of light transmittance, degradability, biocompatibility, anti-inflammatory activity, cell proliferation, and stemness gene expression.

Results : Our data indicated that the unraveling of the helical structure into a more random globular state is accompanied by an increase in the cross-linking index of AM samples. The biological tissue materials cross-linked with carbodiimide for longer time possessed large nanofiber diameter and exhibited rough texture. In addition, the enhanced LEC growth and increased p63 and ABCG2 gene expressions were significantly noted on the AM samples with greater cross-linking degree.

Discussion : Carbodiimide cross-linked AM can potentially serve as an artificial corneal epithelial stem cell niche in ocular surface wound healing. Marked increases in the water content, light transmittance, and resistance to enzymatic degradation were found, probably due to collagen fibril aggregation in biological tissues. All the test AM materials were not toxic to the human corneal epithelial cell cultures and retained anti-inflammatory activity, indicating the safety of carbodiimide.

Conclusions : The findings suggest that a specific limbal epithelial stem cell-biomaterial interaction may occur in response to biophysical cue such as nanostructure of carbodiimide cross-linked AM matrix.

16:30-16:40

S-7

合併使用甲殼素塗佈導管與脂肪幹細胞誘導之神經球細胞來促進坐骨神經功能性恢復
Functional recoveries of sciatic nerve regeneration by combining chitosan-coated conduit and neurosphere cells induced from adipose-derived stem cells

薛元毓^{1,2} 張雅茹^{3,4} 黃子捷⁵ 范詩辰⁶ 王鐸翔¹ 陳家進^{7,8} 吳佳慶^{3,4,7,8} 林聖哲¹
國立成功大學附設醫院整形外科¹ 臨床醫學研究所² 細胞生物暨解剖研究所³
基礎醫學研究所⁴ 職能治療系⁵ 生物醫學研究所⁷ 創新醫材中心⁸ 義大醫院職能治療系⁶

Introduction : Regeneration of peripheral nerve occurs but the results are suboptimal, especially when the nerve gap is encountered. Current advances in biomaterial and tissue engineering have been developed to bridge the gap with various biosynthetic conduits or cell-based therapy. In this study, we use combination of chitosan coated conduit with induced neural progenitor cell from human adipose stem cell to facilitate rat sciatic nerve regeneration.

Materials and Methods : The human ASC from healthy donors are obtained with informed consent and approved according to the procedures of the institutional review board. P3 ASCs are seeded onto chitosan coated surface to form free floating neurospheres spontaneously for 2 days. We use 200~250 g S-D rat to develop the model of sciatic nerve transection with 1-cm critical nerve gap, which are randomly assigned to five groups: Group1-sham operation(Sham); Group2-silicon neural tube(S); Group3-silicon neural tube seeded with chitosan(C); Group4-silicon tube plus induced NPCs(S+N); Group5-chitosan tube plus induced NPCs(C+N). The dynamic gait analysis is evaluated at 1, 3 and 6 weeks after surgery. The rat is sacrificed at 6 weeks after surgery for evaluation of the regenerated nerve and muscle using immunohistochemic staining.

Results : By using a single treatment of chitosan-coated conduit or neurosphere cell therapy, the nerve gap was partially recovered after 6 weeks of surgery. Substantial improvements in nerve regeneration were achieved by combining neurosphere cells and chitosan-coated conduit based on the increase of myelinated axons density and myelin thickness, gastrocnemius muscle weight and muscle fiber diameter, and step and stride lengths from gait analysis. High expressions of interleukin-1 β and leukotriene B4 receptor 1 in the intra-neural scarring caused by using silicone conduits revealed that the inflammatory mechanism could be inhibited when the conduit is coated with chitosan.

Discussion : We developed a cell-assisted chitosan-coated conduit to bridge a 10-mm gap in the rat sciatic nerve transection model. In the early recovery period, 6 weeks after surgery, beneficial effects that promoted axon remyelination, reduced intra-neural scarring, enhanced the re-innervation of the effector muscle, and improved overall gait were observed. These functional improvements may have resulted from an anti-inflammatory effect involving the inhibition of IL1- β and leukotriene signaling.

Conclusion : This study demonstrated that the chitosan-coated surface performs multiple functions that can be used to induce neurosphere cells from ASCs and to facilitate nerve regeneration in combination with a cells-assisted coated conduit, giving great potential for clinical application to autologous cell-based therapies for patients with peripheral nerve injury.

第六屆第一次會員大會

台灣再生醫學學會第五屆理、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、蔡清霖
- 理事 王至弘、王兆麟、方旭偉、江清泉、侯連團、
張至宏、黃玲惠、楊俊佑、楊榮森、嚴孟祿
- 候補理事 林高田、張恒雄、陳文哲、陳志華、陳英和
- 常務監事 黃義侑
- 監事 林文澧、林泰元、孫瑞昇、陳敏慧
- 候補監事 顏伶汝

第六屆第一次會員大會議程

時間：民國103年2月22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. 通過年度工作計劃

2. 通過年度經費收支、決算表

五、臨時動議

六、選舉第六屆理監事

七、散會

102年度工作報告

理事會報告

- 一、召開理監事會議計四次。
- 二、會員實際人數一百七十二人。

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。

103年工作計劃

- 一、招收會員
- 二、舉行四次理監事會議。
- 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 102 年 1 月 1 日至 102 年 12 月 31 日止

科 款	項 目	科 目	決算數	預算數	決算與預算比較數		說 明
					增 加	減 少	
1		本會收入	391,621	630,000			
	1	會費收入	59,000	170,000		111,000	含入會費、常年會費
	2	捐款收入	330,000	450,000		120,000	廣告攤位收入及贊助會員捐款等
	3	利息收入	2,621	10,000		7,379	郵局、銀行利息
2		本會支出	388,764	630,000			
	1	人事費	173,000	170,000	3,000		員工及加班費
	2	文具	1,068	5,000		3,932	
	2	印刷費	12,922	70,000		57,078	
	3	郵電費	2,152	5,000		2,848	
	4	雜項	6,311	10,000		3,689	
3	1	業務費	27,376	70,000		42,624	召開監事會及辦理相關研討會所需之費用
	2	會議費	42,000	80,000		38,000	
	3	交通費	10,500	20,000		9,500	
		其他業務費	105,435	200,000		94,565	召開 2 月份年會
		提撥基金	8,000	31,500		23,500	
		本期結餘	2,857				

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 103 年 1 月 1 日至 103 年 12 月 31 日止

科 款	項 目	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
					增 加	減 少	
1	1	本會收入	600,000	630,000			
	2	會費收入	140,000	170,000		30,000	含入會、費常年會費
	3	捐款收入	450,000	450,000			廣告攤位收入及贊助會員捐款等
		利息收入	10,000	10,000			郵局銀行利息
2	1	本會支出	600,000	630,000			
	2	人事費	180,000	170,000	10,000		員工薪資及加班費
	1	文具	5,000	5,000			
	2	印刷費	50,000	70,000		20,000	
	3	郵電費	5,000	5,000			
	4	雜項	10,000	10,000			
	3	業務費	40,000	70,000		30,000	召開理事會及辦理相關研討會所需之費用
	2	會議費	60,000	80,000		20,000	
	3	交通費	20,000	20,000			
		其他業務費	200,000	200,000			
		提撥基金	30,000	31,500		1,500	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 102 年 1 月 1 日至 102 年 12 月 31 日止

科目名稱	收 入		支 出	
	金額	科目名稱	金額	金額
上期結餘	\$1,693,897	本期支出	\$388,764	
本期收入	\$391,621	本期結餘	\$1,696,754	
合計	\$2,085,518	合計	\$2,085,518	

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 102 年 1 月 1 日至 102 年 12 月 31 日止

科目	資 產		負 債	
	金額	科目	金額	金額
庫存現金	\$1,696,754	累計基金	\$104,000	
--	--	本期結餘	\$1,592,754	
合計	\$1,696,754	合計	\$1,696,754	

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	郭兆瑩
007	林峰輝	037	施庭芳	068	黃國淵	098	陳學明
008	林文澧	038	侯連團	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						
117	蘇正堯	137	徐新生	157	蔡宗廷	177	賴文福
118	楊世偉	138	許文明	158	羅文政	178	施子弼
119	林偉彭	139	黃鶴翔	159	王德原	179	黃鼎鈞
120	謝豐舟	140	陳偉勵	160	賴志毅	180	陳宣佑
121	方紀宇	141	劉席璋	161	吳佳慶	181	邵宏仁
122	蘇慶華	142	李冠瑢	162	沈延盛	182	徐永康
123	曾育弘	143	胡育誠	163	李一麟	183	賴瑞陽
124	林佐文	144	黃維超	164	何美泠	184	薛元毓
125	史 中	145	陳安泰	165	楊宗霖	185	施明光
126	鄭乃禎	146	謝清河	166	吳坤佶	186	曾效參
127	謝式洲	147	彭慶安	167	趙本秀	187	趙崧筌
128	蘇鴻麟	148	劉滄梧	168	鄭明德	188	黃柏誠
129	曾清秀	149	薛敬和	169	李源芳	189	彭凱彥
130	劉百栓	150	林毅誠	170	嚴孟祿	190	李奎璋
131	唐逸文	151	顏君哲	171	顏伶汝		
132	王清正	152	陳江山	172	林泰元		
133	王盈錦	153	侯君翰	173	陳尹愷		
134	吳信志	154	吳俊昇	174	許元銘		
135	簡雄飛	155	廖振焜	175	鄭有仁		
136	高國慶	156	傅再生	176	侯添財		

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		證 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)：											
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：