活動預告:

謝豐舟教授回顧展3D導覽

歡迎上線參觀!!!!

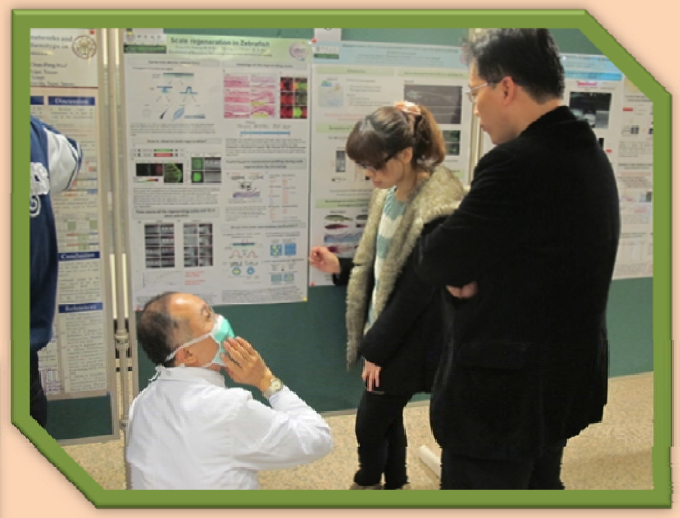
http://www.mc.ntu.edu.tw/alualu/3D.htm

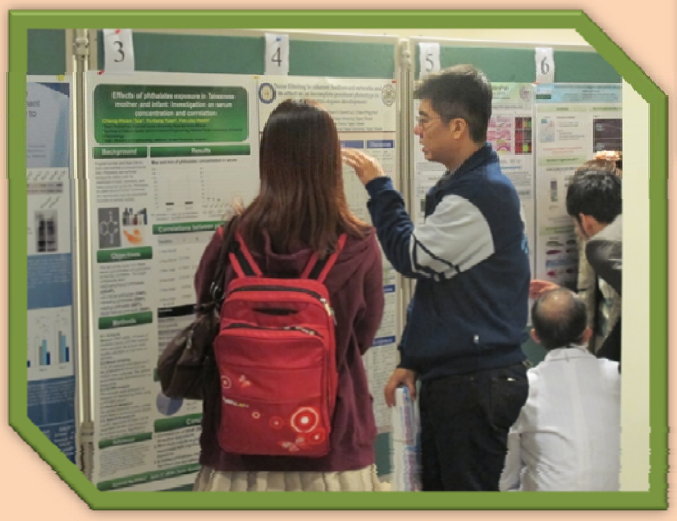
活動花絮:

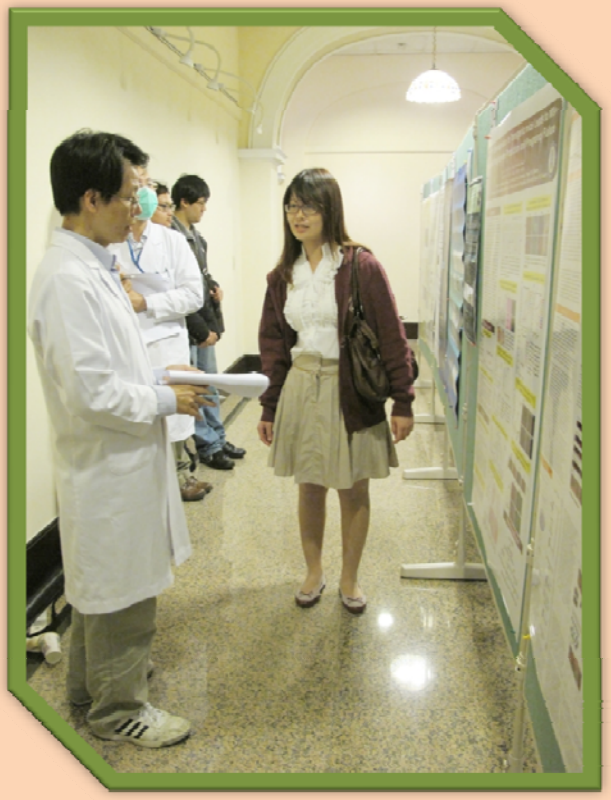
年終發育生物學與再生醫學研究海報研討會暨謝豐舟教授歡送茶會

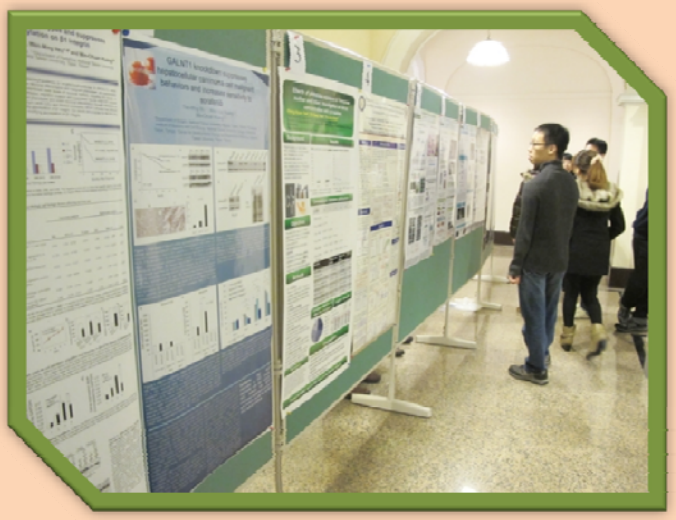
時間:2011年 12月09日

地點: 台大醫學院人文館









對於海報內容討論熱烈

活動花絮:海報得獎者

財團法人謝伯潛醫學教育基金會獎　　得獎人:台大醫學院解剖所 博班 周志行

β-1,4-galactosyltransferase III enhances malignant phenotypes and suppresses neuronal differentiation by modifying glycosylation on β1 integrin

Hsiu-Hao Chang[1,4]*, Chia-Hua Chen[1]*, Chih-Hsing Chou[2], Fon-Jou Hsieh[3], Wen-Ming Hsu[1,4]# and Min-Chuan Huang[1]#

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The role of glycosyltransferases in neural tumorgenesis and differentiation is unknown. Here we reported a galactosyltransferase β-1,4-galactosyltransferase Ⅲ (B4GALT3), which efficiently catalyzes the synthesis of the first N-acetyllactosamine unit. In order to investigate the role of B4GALT3 in neuronal differentiation and malignant phenotypes, we overexpressed B4GALT3 in human SH-SY5Y cells. After retinoic acid induced neuronal differentiation, B4GALT3 transfectants expressed lower levels of neuronal markers, including βⅢ-tubulin, α-internexin, and NF-H, compared with mock transfectants. Following overexpression of B4GALT3 enhanced cell adhesion, migration invasion and suppressed neurite outgrowth induced by serum starvation on collagen Ⅳ, fibronectin, and laminin, which suggests that B4GALT3 may promote malignant phenotypes and inhibit neuronal differentiation through interaction of extracellular matrix proteins and their receptors. Furthermore, lectin pull-down assays using *Lycopersicon Esculentum Lectin* (LEL) and *Ricinus communis I* (RCA I) showed that different glycosylation patterns of β1 integrin between B4GALT3 and mock transfectants, indicating that B4GALT3 can modify glycosylation of β1 integrin. Our results demonstrate for the first time that B4GALT3 enhances malignant phenotypes and inhibits neurite outgrowth and neuronal differentiation through modifying glycosylation on β1 integrin.

活動花絮:海報得獎者

發育再生中心榮譽主任鍾正明院士獎　得獎人:中央研究院 分生所博班　簡瑜

Cyp11a1 Overexpression in Transgenic mice Leads to Mis-regulated Progesterone Production and Pregnancy Failure

Yu Chien[1,2], Wei-Cheng Cheng, Bon-chu Chung[1,2]
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The *CYP11A1* gene encodes the P450scc, which catalyzes the first step in steroid biosynthesis. *CYP11A1* overexpression is associated with predisposition to breast cancer, endometrial cancer and polycystic ovary syndrome. Some of these patients also suffer from pregnancy problem. To characterize the obstetric and gynecological defects caused by *Cyp11a1* overexpression, we generate *Cyp11a1* transgenic mice using a mouse bacterial artificial chromosome that recapitulates the endogenous expression of *Cyp11a1* gene. We found that the transgenic females had elevated estradiol (E2) levels at metestrus and diestrus and more cell death of corpora lutea at diestrus. The mural granulosa cells possessed more mitotic ratios in preovulatory follicles of transgenic mice. *Cyp11a1* transgenic females displayed reduced pregnancy rate, decreased litter size *in utero*, delayed parturition and no live birth. Less serum progesterone (P4) was produced from transgenic females during early pregnancy. The progesterone level returned to normal afterwards, but its withdrawal at term was delayed. Insufficient progesterone in *Cyp11a1* transgenic females could not support normal implantation and placentation at the correct time, leading to more absorption of embryos. The luteal cells of transgenic ovaries appeared foamy and accumulated numerous lipid droplets in the cytoplasm during early pregnancy. The expressions of genes related to steroidogenesis and cholesterol homeostasis in transgenic ovaries were diminished during maturation process of corpora lutea. Prostaglandin E2 signaling was raised for preventing cell death of transgenic corpora lutea at immature stage. Taken these findings together, we conclude that overexpression of *Cyp11a1* disrupts the normal development of corpus luteum then leads to progesterone insufficiency during early pregnancy. Therefore *Cyp11a1* transgenic females can serve as a disease model of recurrent pregnancy failure.

活動花絮:海報得獎者

發育再生中心主任楊偉勛教授獎　　得獎人:台大化學所博班　陳奕丞

Noise filtering in coherent feedforward networks and its effect on an incomplete penetrant phenotype in *Caenorhabditis elegans* development

Yi-Chen Chen[1,2] , Yi-Chun Wu[3] , Chun-Yi David Lu[1], Chao-Ping Hsu[2]
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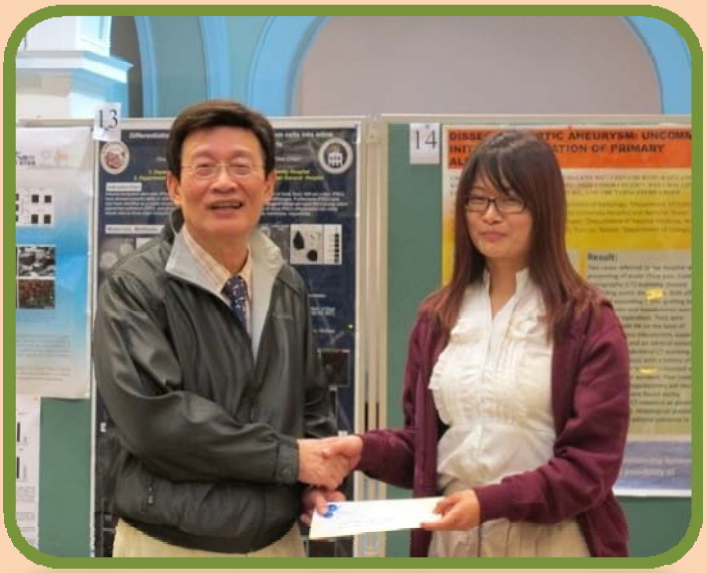
Gene expression noise is ubiquitous in cells. In the development of multicellular organisms, a genetic regulatory network must be able to cope with the fluctuations in the gene expression level so that a final phenotype is nearly the same in different individuals. However, not much is known about the mechanisms that may result in the fluctuations at the gene expression level in multicellular organisms and how the fluctuations may be buffered in a way that leads to a ubiquitous output in different individuals. In the present work, we study the effect of gene expression fluctuations on the timing control of cell migration in the wild-type and mutant individuals of the model organism *C. elegans*. Specifically, we examine a pair of somatic cells termed distal tip cells (DTCs), which guide the migration direction of the gonadal arms during larval development. Previous molecular and genetic data have established a network that consists of multiple interlinked feedforward loops required for the timing control of DTC ventral to dorsal turning at the third larval stage. Mutations in the hub of this genetic network cause an incomplete penetrant turning defect, despite the fact that mutants were of an identical genetic background and grew under the same condition. To see whether the noise filtering capacity might be disrupted in these mutants, we create a dynamic model, in which we randomly introduce noisy spikes into the OFF or ON state of the input signals, regulators of the network, and simulate the output signal, a downstream effector of the network. In addition, we have determined a Boolean logic in the genetic network according to the noise filtering effect to reproduce experimental phenotype variations. We find that the simulated results fit well to most of the experimentally observed phenotypes. We demonstrate that each incomplete penetrant phenotype observed in mutants can be attributed to a specific noisy input. Our results suggest that noises in the input signals at the gene expression level can contribute to an incomplete penetrant mutant phenotype in the timing of cell migration in a population.

活動花絮:

財團法人謝伯潛醫學教育基金會獎

得獎人:台大醫學院解剖所 博班 周志行



發育再生中心榮譽主任鍾正明院士獎

得獎人:中央研究院 分生所博班　簡瑜

發育再生中心主任楊偉勛教授獎

得獎人:台大化學所博班　陳奕丞

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楊偉勛教授主持

杜永光教授

胡海國教授

蕭水銀教授

蔡世峰教授

葉素玲教授

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左:孫維仁教授
右:鄭文芳教授

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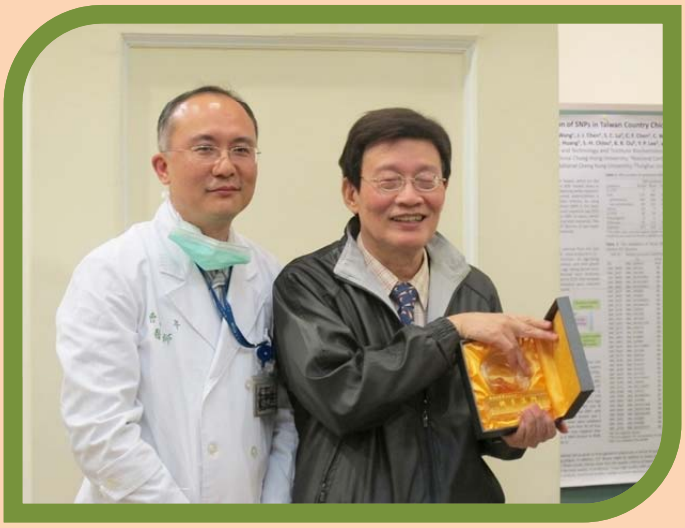

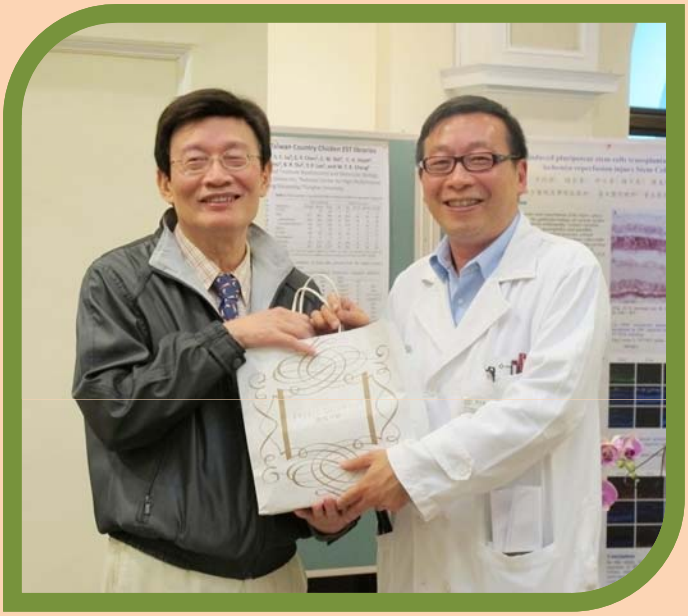

贈送禮物給這位台大傳奇經典人物-謝豐舟教授

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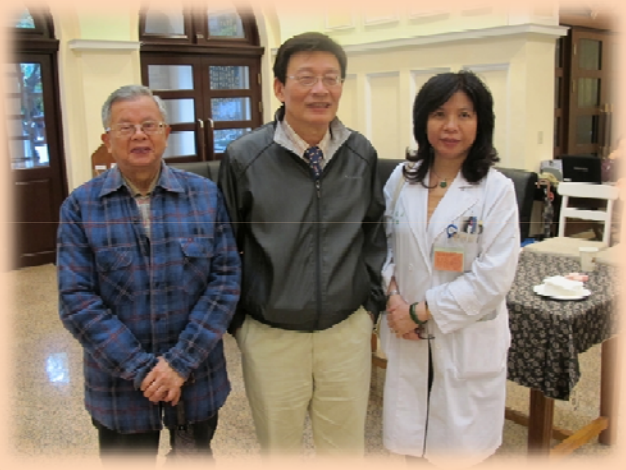

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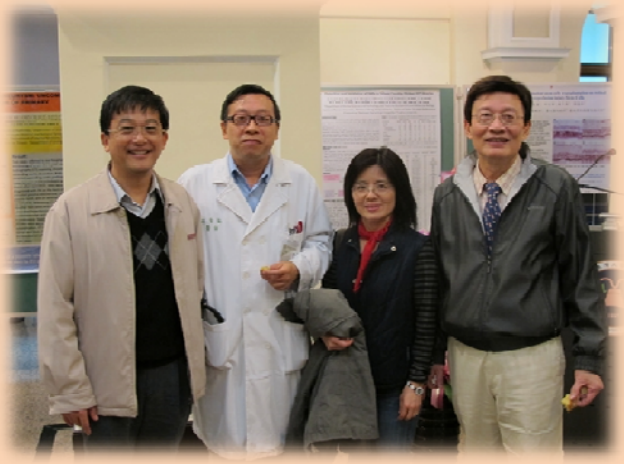

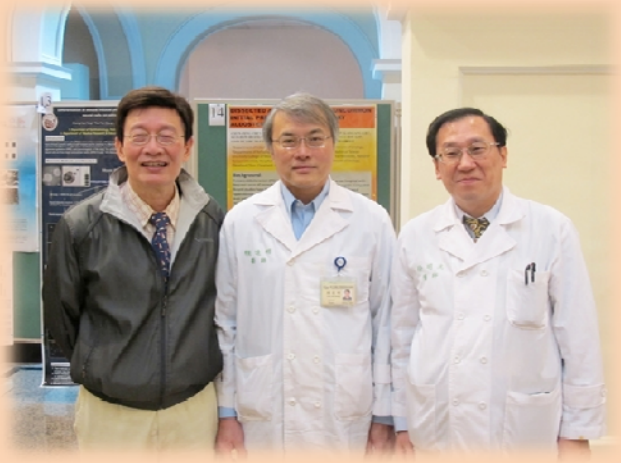

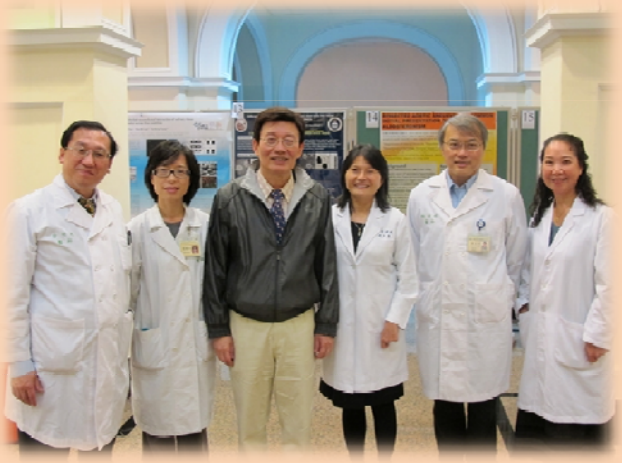

活動花絮:

左: 來自奧地利鋼琴家-劉美貞
右: 陳弘觀-藥理學博班

活動花絮:

這些年輕學子們想對您訴說: 感謝您40年載 無私的付出