

大学院特別講義のご案内

日時： 12月12日(月) 16:30~18:00

場所： 弓倉記念ホール(F棟5階)

講師： Professor. Yang Chai

(Center for Craniofacial Molecular Biology, Ostrow School of Dentistry, University of Southern California)

演題： 「Craniofacial mesenchymal stem cells in bone tissue homeostasis and repair」

- **要旨：** Bone tissue undergoes constant turnover supported by stem cells. Recent studies showed that perivascular mesenchymal stem cells (MSCs) contribute to the turnover of long bones. Craniofacial bones are flat bones derived from a different embryonic origin than the long bones. We have recently identified *Gli1*⁺ cells within the suture mesenchyme as the major MSC population for craniofacial bones. They are not associated with vasculature, give rise to all craniofacial bones in the adult and are activated during injury repair. *Gli1*⁺ cells are typical MSCs *in vitro*. Ablation of *Gli1*⁺ cells leads to craniosynostosis and arrest of skull growth, indicating these cells are an indispensable stem cell population. *Twist1*^{+/-} mice with craniosynostosis show reduced *Gli1*⁺ MSCs in sutures, suggesting that craniosynostosis may result from diminished suture stem cells. Craniofacial sutures provide a unique niche for MSCs for craniofacial bone homeostasis. During calvarial bone repair, the calvarial sutures possess much stronger regeneration capability than the non-suture areas. The healing rate of the calvarial bone is inversely related to the suture-injury distance. Any injury closer to the suture heals faster. After complete removal, the sagittal suture can regenerate and restore to normal organization within 6 weeks. *Gli1*⁺ cells within the suture mesenchyme are the cellular source for the injury repair. These results suggest that calvarial bone healing is not an evenly distributed event on the calvarial surface. The suture is the origin of its capacity for regeneration. Our study has important implications for the preservation of suture structures during calvarial surgery and can also help us to design new approaches for repairing calvarial malformations and defects. Supported by R37 DE012711 and R01 DE025221, NIDCR, NIH