生物科技學系
分子醫學與生物工程所
電話：03-5712121 轉 59725
E-mail : chao7@nctu.edu.tw
實驗室 : 微小型核醣核酸及腫瘤幹細胞實驗室

趙 啟 宏 助理教授

研究興趣

我的研究方向，主要在闡明腫瘤微環境影響腫瘤幹細胞的表觀遺傳學特徵與代謝途徑變異的分子機制。本實驗室先前的研究指出，腫瘤微環境中的訊號分子可透過調節微型核糖核酸的表現而影響腫瘤的發展與腫瘤幹細胞特性的擴張。由於這些微型核糖核酸在調節腫瘤代謝與腫瘤幹細胞特性上扮演重要角色，因此我的研究方向亦包含了利用腺相關病毒來進行微型核糖核酸的基因傳遞，以作為腫瘤治療策略的開發。本實驗室相關的研究，將有助於了解表觀遺傳學變異，代謝途徑轉變與腫瘤幹細胞特性三者間的關聯，而藉由鑑定出此關聯性的關鍵分子與其作用機轉將有助於我們發展出有效的腫瘤治療或預防策略。

本實驗室正進行中的研究計畫如下：

- 探討microRNA-200c所調節的代謝途徑變異在三陰性乳癌發展中所扮演的生物意義。
- P53突變在三陰性乳癌中所引起的代謝途徑變異及其臨床意義。

Assistant Professor ,Department of Biological Science and Technology,
Institute of Bioinformatics and Systems Biology
TEL: 886-3-5712121 ext. 59725
E-mail: chao7@nctu.edu.tw
Lab: Laboratory of MicroRNA and Cancer Stem Cells

Chi-Hong Chao, Ph.D.

Research Interests

My current research is focused on revealing critical molecular mechanisms in which the tumor microenvironment regulates the epigenetic status and metabolic alternation of breast cancer stem cells. Our data show that aberrant stimuli from tumor microenvironments could lead to cancer progression and cancer stem cell expansion through modulating microRNA expression. Since these microRNAs play an important role in controlling metabolism and cancer stem cell properties, my research interests also include developing novel target therapy by using Adeno- associated virus (AAV)-mediated microRNA delivery. Future studies in this field are expected to open a new avenue by elucidating the link between epigenetics, metabolism and cancer stem cells. I believe that identifying the key regulatory mechanisms or components which promote cancer stem cells will unravel important therapeutic targets for eradicating the genesis of cancer and prevent cancer progression. Ongoing projects are listed below:

- **The biological roles of microRNA-200c regulated metabolic alternations in triple-negative breast cancer (TNBC) progression**

TNBC is the most aggressive breast cancer type with worst survival rate, and decreased expression of microRNA-200c has been observed in more than 70% of TNBC patients, which indicates miR-200c-deficiency-induced epithelial-mesenchymal transition (EMT) and enhanced cancer stemness may play a critical role in TNBC progression. In viewing that altered metabolic flux such as aerobic glycolysis facilitates tumor formation and metastasis, and lipid metabolism has been linked to tumor initiation, metastasis and resistance to chemotherapy through regulating stem cell properties, we are interested to know (i) whether miR-200c harbors the ability to regulate cellular metabolisms, and (ii) whether miR-200c-deficiency in TNBc leads to metabolic alternations, which in turn contribute to TNBC progression.

- **P53 mutants-mediated metabolic alternations in TNBC and its clinical significance**

P53 mutations can be found in more than 80% of TNBC patients, and this high mutation rate suggests a critical role of p53 in TNBC formation. In this project, we aim to elucidate the effect of p53 mutations in regulating metabolic flux in TNBC cells, and further clarify the biological roles of these metabolic alterations in TNBC progression. Since miR-200c is a direct target of p53, we also like to investigate whether miR-200c expression is able to counteract the metabolic effects exerted by p53 mutants. This study will pave the way for gene therapy targeting cancers with p53 mutations.