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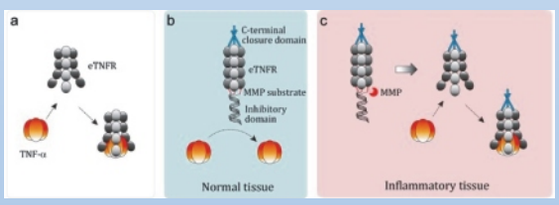
研究興趣

本實驗室運用免疫學的知識與概念，來研究人類的疾病，並且嘗試發展新式治療藥物。目前有三個主要的研究方向。

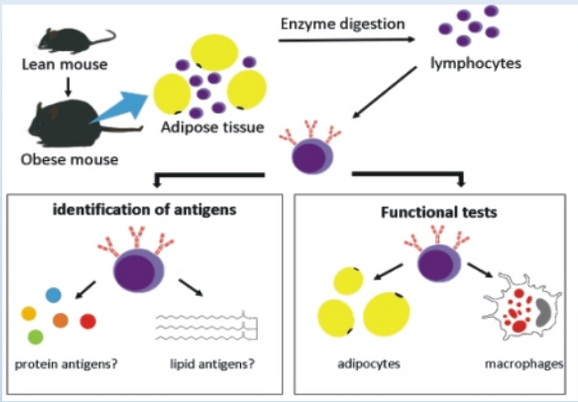
• 發展新式治療型蛋白藥物

現行臨床上已有許多治療用的重組蛋白(例如抗體或受體嵌合蛋白)，然而這類重組蛋白藥物常造成的全身性副作用。為了解決這個問題，我們利用在病灶組織中大量表現的蛋白酶作為活化藥物的機制，開發不會影響正常組織的新型重組蛋白藥物。我們將不同的蛋白質序列連結蛋白酶的受質序列，接合到抗體或受體嵌合蛋白的N-端，藉此遮蔽抗體或受體嵌合蛋白的結合位。當此新型重組蛋白藥物進入發炎組織後，利用高度表現的蛋白酶將遮蔽蛋白切除活化後，抗體或受體嵌合蛋白的活性可以完全恢復 (Chen IJ , et al. 2017. Scientific Reports; Lee CJ, et al. 2019. Cytokine)。



• 代謝症候群免疫病理機制探討與治療藥物研究

代謝症候群是能量代謝異常導致的疾病，其病徵包括胰島素抗性、高血糖、高血脂等。目前研究顯示代謝症候群與肥胖強烈相關，而且免疫細胞 (包括T細胞與B細胞)會浸潤到肥胖的脂肪組織內導致發炎，並且造成胰島素抗性。為了瞭解B細胞在代謝症候群扮演的角色，我們從肥胖小鼠的脂肪組織分離出B細胞，建立融合瘤細胞，希望能作為研究代謝症候群致病機制的有用工具，並藉此發展出有效的治療方法。



• 自體免疫腦下垂體炎病理機制

自體免疫腦下垂體炎是一種T和B細胞浸潤到腦下垂體的慢性發炎疾病。目前對T和B細胞導致疾病的機制仍不清楚。我們在自體免疫腦下垂體炎的小鼠模式與病人檢體中，都發現浸潤腦下垂體的T和B細胞會在腦下垂體二次活化，產生高量的干擾素-γ (IFN-γ)和介白素-17 (IL-17)，並進行原位增生，持續產生自體免疫性的T與B細胞，這可以解釋腦下垂體持續慢性發炎的原因(Lin HH, et al. 2017. Scientific Reports)。

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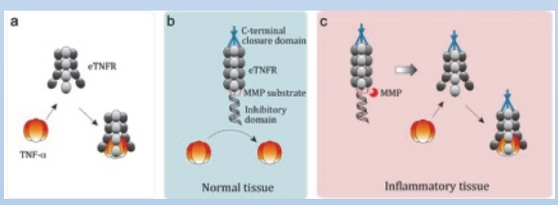
Research Interests

In our laboratory we apply the knowledge and concepts of immunology to study human diseases and develop new therapeutic drugs. There are currently three main research directions.

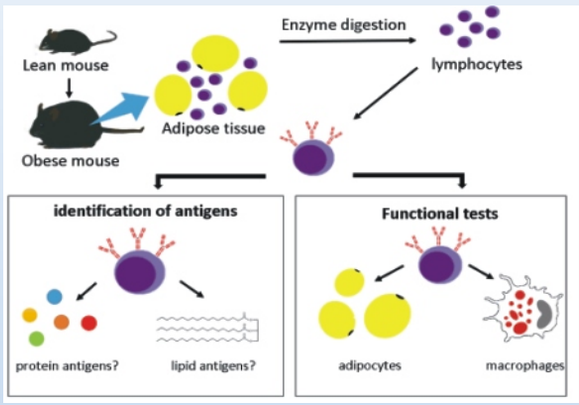
• Developments of novel therapeutic protein drugs

Many recombinant proteins (antibodies and chimeric receptor proteins) are in clinical use, but these protein drugs often cause adverse effects. To address this issue, we linked a masking protein domain, through a substrate sequence, to the N-terminus of the antibody or chimeric receptor protein, thereby masking the binding site of the antibody or receptor. The antibody or chimeric receptor protein can be revived, upon cleavage on the substrate sequence to release the masking protein, by proteases highly expressed in the inflammatory tissues (Chen IJ, et al. 2017. *Scientific Reports*. Lee CJ, *et al*. 2019. *Cytokine*).



• Investigation of the pathogenesis of metabolic syndrome

Metabolic syndrome is a complex disease associated with obesity, manifesting insulin resistance, hyperglycemia and hyper-lipidemia, among others. It is also known that immune cells (including T and B cells) infiltrate obese adipose tissue, leading to inflammation and insulin resistance. We isolated B cells from adipose tissues of obese mice and generated hybridomas. These hybridomas may serve as a useful research tool for studying the roles of antibody and B cells in metabolic syndrome and for developing potential therapies.



• Investigation of the pathogenesis of autoimmune hypophysitis

Autoimmune hypophysitis is a chronic inflammatory disease mediated by T and B cell infiltration into the pituitary. The pathogenesis autoimmune hypophysitis remains unclear. We found that pituitary-infiltrating T and B cells receive secondary activation *in situ*, in both a mouse model and in human patients. These T and B cells undergone proliferation and produced heightened cytokines IFN-γ and IL-17. These findings provide a plausible mechanism to explain how chronic inflammation in the pituitary gland is sustained (Lin HH, *et al*. 2017. *Scientific Reports*).