

花蓮慈濟醫院研究部

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研究簡介

A. The major research interest in our laboratory has been focused on the mechanisms of INFLAMMATION by using different animal models.

My research interest has been always focused on the mechanisms of inflammation. By using different aspects of knowledge and techniques, such as transgenic and stem cell technology, our team specialized in creating different unique and specific animal models to answer the questions sometimes hard to have a clue from other models.

B. The inflammatory neuropathic injury related research works and publication. We formally proved the contribution of inflammation to neuropathic pain by using immune compromised mice. We also identified that *leukocyte derived endogenous opioids are* native negative feedback system toward inflammation derived pain. Then we focused on the chemokines CCL5, one of the major chemokines in inflammation, and found CCL5 is critical to the development of neuropathic pain and could be a future target for drug development (**Selected as cover in J of Pain**).

C. The A2AAR and ischemia reperfusion, inflammatory mechanisms related studies. We found the possible major target for A2AAR mediated protection is allocated at the very early stages of reperfusion, such as CD4+ TC, NKT and DCs. We also found that A2AAR would suppress the platelets release its content, including RANTES, upon activation. Further, both A2AAR and released RANTES (CCL5) is essential to the regulatory T lymphocytes production, but in opposite direction for each.

D. The platelets and ischemia reperfusion derived inflammation related research works

Interestingly, the excessive ex vivo produced HPSC platelets demonstrated anti-inflammation characteristics, which has been proved in many inflammatory models in our lab, including sepsis, liver IR, acute kidney injury, burn injury, stroke models etc.

RESEARCH INTERESTS IN DYJ LABORATORY Transgenic techniques Immunological techniques Stem cell techniques Animal models **Inflammation** DM, obesity

Cancer

DM, obesity

計畫與經費來源

	研究計畫(計畫編號)	主持人	執行期間	執行單位
1	第三沉默調控因子(SIRT3)於肝臟缺血在灌注傷害中之角色探討(MOST 105-2314-B-303-021-MY2)	戴元基	2016/08/01~2019/07/31	科技部
2	第三沉默因子對於小鼠敗血症模式及其巨噬細胞功能的影響(TCRD106-56)	戴元基	2017/01/01~2019/12/31	院內
3	由細胞和分子層面探討沉默信息調節蛋白3在小鼠神經性疼痛模式中所扮演之角色(105-2314-B-182A-015-)	戴元基	2016/08/01~ 2017/07/31	科技部
4	RANTES訊息傳遞路徑在肥胖發展及相關心血管和代謝疾病中所扮演的角色—(子計畫二)肥胖相關心血管功能異常的病理機制中RANTES 訊號扮演之角色(3/3)(104-2320-B-182-001-)	戴元基	2015/08/01~ 2016/07/31	科技部
5	RANTES訊息傳遞路徑在肥胖發展及相關心血管和代謝疾病中所扮演的角色—(子計畫三)RANTES在肥胖誘導之代謝異常所扮演的免疫病理角色探討(2/3)(103-2320-B-182A-001-)	戴元基	2014/08/01~ 2015/07/31	科技部
6	以IL-17為新穎標的探討黃芩類黃酮在藥物誘發肝臟損傷之保護效果(103-2314-B-182-046-MY2)	戴元基	2014/08/01~ 2016/07/31	科技部
7	利用基因剔除鼠探討P-選擇素糖蛋白配體-1對小鼠神經性疼痛之影響(102-2314-B-182A-105-MY3)	戴元基	2013/08/01~ 2016/07/31	科技部
8	腺甘酸A2A接受器調控再灌注發炎時骨髓細胞之交互作用研究(102-2628-B-182A-011-MY3)	戴元基	2013/08/01~ 2016/07/31	科技部
9	利用基因剔除鼠和骨髓移殖方法探討白細胞介素-17對小鼠神經性疼痛之影響(101-2314-B-182A-011-)	戴元基	2012/08/01~ 2013/07/31	科技部
10	探討石斑木成份藉由PI3K調控發炎免疫反應機轉及其動物肝臟缺血性再灌流傷害之研究(101-2320-B-182-019-MY3)	戴元基	2012/08/01~ 2015/07/31	科技部
11	趨化激素受體CCR1和CCR5在乙醯氨基酚(Acetaminophen)誘發小鼠肝損傷中之角色探討(101-2314-B-182-082-)	戴元基	2012/08/01~ 2013/07/31	科技部

研究成果

- 1. Lee CM, Peng HH, Yang P, Liou JT, Liao CC, Day YJ: C-C Chemokine Ligand-5 is critical for facilitating macrophage infiltration in the early phase of liver ischemia/reperfusion injury. Sci Rep. 16;7(1):3698. 2017 (IF= 5.578 5/57, 8.77%, Multidisciplinary Sciences)
- 2. Day,Y.J.; Chen,K.H.; Chen,Y.L.; Huang,T.H.; Sung,P.H.; Lee,F.Y.; Chen,C.H.; Chai,H.T.; Yin,T.C.; Chiang,H.J.; Chung,S.Y.; Chang,H.W.; Yip,H.K.: Preactivated and Disaggregated Shape-Changed Platelets Protected Against Acute Respiratory Distress Syndrome Complicated by Sepsis Through Inflammation Suppression. Shock 46(5):575-586, 2016 (IF= 3.048 33/199, 16.58%, Surgery).
- 3. Chou, A.H., C.M.Lee, C.Y.Chen, J.T.Liou, F.C.Liu, Y.L.Chen, and Y.J.Day*. Hippocampal transcriptional dysregulation after renal ischemia and reperfusion. *Brain Res.* 1582:197-210. 2014 (IF= 2.828, 134/251, 53.38%, Neuroscience)
- 4. Day, Y.J*., J.T.Liou, C.M.Lee, Y.C.Lin, C.C.Mao, A.H.Chou, C.C.Liao, and H.C.Lee. 2014. Lack of interleukin-17 leads to a modulated microenvironment and amelioration of mechanical hypersensitivity after peripheral nerve injury in mice. Pain 155:1293-1302. 2014 (IF= 6.428, 1/28, 3.57% Anesthesiology)
- 5. Cekic, C., D. Sag, Y. J. Day, and J. Linden. Extracellular adenosine regulates naive T cell development and peripheral maintenance. J. Exp. Med. 210:2693-2706. 2014 (IF= 14.102, 2/112, 1.78% Medicine, research & experimental)
- 6. Liou, J.T., C.M.Lee, Y.C.Lin, C.Y.Chen, C.C.Liao, H.C.Lee, and Y.J.Day*. P-selectin is required for neutrophils and macrophage infiltration into injured site and contributes to generation of behavioral hypersensitivity following peripheral nerve injury in mice. *Pain* 154:2150-2159. 2013 (IF= 6.428, 1/28, 3.57% Anesthesiology)
- 7. Liou, J.T., C.C.Mao, S.D.Ching-Wah, F.C.Liu, Y.S.Lai, J.C.Li, and Y.J.Day*. Peritoneal administration of Met-RANTES attenuates inflammatory and nociceptive responses in a murine neuropathic pain model. *J.Pain* 14:24-35. 2013 (IF= 4.245, 9/192, 4.68% clinical neurology)
- 8. Liou, J.T., H.B. Yuan, C.C. Mao, Y.S. Lai, and Y.J. Day*. Absence of C-C motif chemokine ligand 5 in mice leads to decreased local macrophage recruitment and behavioral hypersensitivity in a murine neuropathic pain model. *Pain* 153:1283-1291. 2012 (IF= 6.428, 1/28, 3.57%) **Anesthesiology**)
- 9. Liou, J.T., F.C.Liu, C.C.Mao, Y.S.Lai, and Y.J.Day*. Inflammation confers dual effects on nociceptive processing in chronic neuropathic pain model. *Anesthesiology* 114:660-672. 2011 (IF= 5.302, 2/28,7.14% Anesthesiology)

Patent Approved:

Therapeutic methods of preactivated and disaggregated shape changed platelets in acute and emergent inflammatory diseases. Aug, 2012.