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• 中文關鍵字	雙相情感障礙症；免疫調節；細胞激素；；；；；
• 英文關鍵字	bipolar disorder；immunomodulation；cytokine；；；；；
• 中文摘要	重大精神病如雙相情感障礙症(Bipolar disorder, 以下簡稱躁鬱症)，重鬱症(Major depression) 以及精神分裂症(Schizophrenic disorder)其確切病因目前均仍未知，但已經有許多的研究顯示上述精神疾病患者的免疫機能有異於常人之處，尤其是其免疫調節因子的細胞激素以及其受體的變化與症狀有某種程度的關聯性。 本研究主持人已對於躁鬱症躁期的研究發現：躁症病患有細胞免疫功能(cell-mediated immunity)的亢進現象，其血清中介白質素-2 受體(soluble interleukin-2 receptor, sIL-2R)顯著升高，並與躁症狀嚴重度呈正相關，其他免疫調節因子的變化初步研究結果(如 血清中 sIL-6R 濃度沒有改變)，推測躁鬱症躁期的免疫變化機轉可能異於輕型精神疾病以及其他精神病（包括精神分裂症與重鬱症）。希望能進一步改良先前研究方法的諸多缺失，深入探討躁鬱症病患於急性躁症期間更多免疫調節因子的變化，主要研究目的爲:釐清與躁期有關之精神生理免疫變化與急性躁症臨床特徵與免疫因子的關聯性。 本研究將收集 34 位確定診斷爲躁鬱症之急性躁期病患，經病患同意後分別於急性期(楊氏躁症量表>26 分)、初緩解期(楊氏躁症量表<12 分)以及完全恢復期(持續八週的緩解期-YMRS<5)抽血檢查，再與性別以及年齡爲控制因素，一對一選擇健康之正常對照組抽血進行比較。 結果:預估本研究 1 年期間收集 34 位個案加以研究，並藉由較大樣本進行統計分析其他各臨床變項，以及藥物對於免疫調節因子的影響。CRP,sTNF-R1 在三種不同情緒狀態下均顯著高於正常對照組;sTNF-R1 則在緩解期顯著升高。瘦體素(leptin)在初緩解期有明顯高於常人。 結論:躁期的確伴隨免疫調節機轉的活化,躁鬱症病患可能伴隨慢性常期的發炎反應，這發炎反應可能與易罹心血管疾病或肥胖有關
• 英文摘要	Background:C-reactive protein (CRP) and the adipose tissue secretion of pro-inflammatory cytokines such as tumour necrosis

factor(TNF)-α and its soluble receptors (sTNF-R1& sTNF-R2) are proinflammatory markers that run in parallel with obesity and cardiovascular risk factors (Mendall et al 1997). It is well known that patients with bipolar disorder are vulnerable to circulatory morbidity and mortality (Tsai et al 2005). The aim of this study is to investigate the immuno-regulatory alternation throughout the manic episode. Method: The plasma levels of leptin, CRP and sTNFR1 in 34 physically healthy patients with bipolar I disorder, manic (DSM-IV) aged 18-45 years were measured in acute mania (YMRS>26), early remission (YMRS<12), and full remission (YMRS<12 for 8+ weeks). The results were compared with age- and sex-matched healthy normal controls. Results: The mean plasma levels of CRP and sTNF-R1 in acute mania, partial remission, and full remission were all significantly higher than that of control subjects.The mean level of sTNF-R1 in early remission was significantly higher than that in either acute mania or full remission. There was no difference in the mean BMI and the plasma leptin levels between each phase of patients and normal controls, except higher leptin in early remission than controls. Conclusion:The present findings provide additional evidence to support that the immunomodulatory systems may be activated during bipolar mania (Tsai et al 1999; 2001). The chronic inflammation, regardless of bodyweight change, may play a role in increasing risk of developing circulatory morbidity. References Mendall MA et al: Relation of serum cytokine concentrations to cardiovascular risk factors and coronary heart disease. Heart 1997; 78:273–277. Tsai SY et al: A retrospective analysis of risk and protective factors for natural death in bipolar disorder. J Clin Psychiatry 2005; 66; 1586-1591 Tsai SY et al: Effects of symptomatic severity on elevation of plasma soluble interleukin-2 receptor in bipolar mania. J Affect Disord 2001; 64:185-193. Tsai SY et al: Activation of indices of cell-mediated immunity in bipolar mania. Biol Psychiatry 1999; 45: 989-994. Acknowledgement This study is supported by research grant from the Department of Health, Taiwan (DOH97-PAB-1002-L).