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• 計畫中文名稱	親皮質素釋放因子和細胞激素之間的神經-內分泌-免疫交互作用對嬰兒點頭痙攣病人的影響		
• 計畫英文名稱	The Neuro-Endocrine-Immune Interaction of CRF and Cytokine in Patients of Infantile Spasm (I)		
• 主管機關	行政院國家科學委員會	• 計畫編號	NSC86-2314-B038-016
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• 中文關鍵字	促腎上腺皮質激素；糖皮素；細胞激素；癲癇；嬰兒點頭痙攣；親皮質素釋放因子		
• 英文關鍵字	Adrenocorticotrophic hormone (ACTH)；Glucocorticoid；Cytokine；Epilepsy；Infantile spasm；Corticotropin releasing factor		
• 中文摘要	<p>嬰兒點頭痙攣(Infantile spasm)是嬰兒時期特有的癲癇。臨床上親腎上腺皮質素(ACTH)及腎上腺糖性類皮質酮(Glucocorticoid)可以極有效的抑制痙攣的發生。基於此臨床上的特徵,過去許多人推論,親皮質素釋放因子過多(CRF- excessive theory)可能是引起嬰兒點頭痙攣的重要致病因子。然而,以前報告並沒有顯示在嬰兒點頭痙攣病人的腦脊髓液中親皮質素釋放因子比正常人高。我們重新檢驗親皮質素釋放因子過多的理論,經由測定 6 位嬰兒點頭痙攣病人血中親皮質素釋放因子的濃度與年齡相近而只有輕微感染的病人(n=12)相比較,發現嬰兒點頭痙攣病人的親皮質素釋放因子確實比感染的病人顯著的(p<0.05)增高。嬰兒點頭痙攣病人的血中親皮質素釋放因子濃度為 0.764.plmin.0.866ng/ml,n=6,而感染病人的為 0.135.plmin.0.076ng/ml,n=12。以 PHA-弓 發淋巴細胞增生在嬰兒點頭痙攣 (525.0.plmin.92.05%,n=3)比在感染的病人(272.7.plmin.51.84%,n=11)有意義的增高(p<0.05)。而且在 PHA-弓 發淋巴細胞增生在嬰兒點頭痙攣比在癲癇病人(Seizure)也是呈現有意義的增加(p<0.01)。此外血清中溶解性介白質-2 受體(sIL-2R)和溶解性介白質-6 受體(sIL-6R)濃度顯示在嬰兒點頭痙攣和癲癇病人並無意義的差異。這些研究結果支持親皮質素釋放因子---過多理論並指出血中高濃度的親皮質素釋放因子表示過多的親皮質素釋放因子對腦神經細胞形成不正當的興奮可能是導致嬰兒點頭痙攣產生的因素。此外增高的確 PHA-弓 發淋巴細胞增生實驗顯示高濃度的血中親皮質素釋放因子可能影響神經-內分泌-免疫功能。</p>		
• 英文摘要	<p>Infantile spasm (IS) is an age-specific seizure syndrome of infancy. Uniquely, the IS seizure respond to hormonal manipulation using adrenocorticotrophic hormone (ACTH) or glucocorticoids. Based upon this clinical feature, a corticotropin-releasing factor (CRF)-excessive theory for the pathogenesis of IS has been proposed and investigated. However, previous reports did not shown any significant change of CRF level in CSF</p>		

of IS patients. We re-examined the CRF-excessive hypothesis by quantifying the CRF level in patients blood sample using EIA assay. Six patients of IS without infection and twelve age-matched patients with minor infection patients (control) were enrolled. The mean age for IS and infection patients is 15.plmin.6 months and 13.5.plmin.1.5 months, respectively. The serum CRF level, but not serum cortisol and ACTH, of IS patient (0.764.plmin.0.866ng/ml, n=6) is significantly higher ($p<0.05$) then control (0.135.plmin.0.076ng/ml, n=12). The phytohemagglutinin (PHA)-induced lymphocyte proliferation in IS (525.0.plmin.92.05%, n=3) is significantly higher ($p<0.05$) than in control (272.7.plmin.51.84%, n=11). Furthermore, PHA-induced lymphocyte proliferation in IS is significantly higher ($p<0.01$) than in seizure (235.5.plmin.65.74%, n=9). However, serum sIL-2R and sIL-6R concentration show no statistical difference between IS and seizure. All these results support the CRF-excess theory and indicate higher CRF expression may contribute to the IS. The elevated PHA-induced lymphocyte proliferation assay demonstrate elevated circulating CRF may influence neuro-endocrine-immune regulation in IS.