• 系統編號	RC8910-0360
• 計畫中文名稱	探討 G1/S 控制點基因與蟹足腫病人致病病因的相關---I.比較病人正常與蟹足腫組織之 G1/S 期控制點基因的表現
• 計畫英文名稱	Involvements of the G1/S Checkpoint Genes in Pathogenesis of Keloid--- I. Comparisons of G1/S Checkpoint Gene Expressions in Paired Normal/Keloid Tissues in the Same Patient
• 主管機關	行政院國家科學委員會
• 計畫編號	NSC88-2314-B038-126
• 執行機構	台北醫學院醫學研究所
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• 使用語言	中文
• 研究人員	李婉若 Lee, Woan-Ruoh
• 中文關鍵字	蟹足腫; 基因表現; 細胞週期蛋白激脢抑制分子; G1/S 期節制點; 細胞增殖; 皮膚疾病; 致病機轉
• 英文關鍵字	Keloid; Gene expression; Cyclin dependent kinase inhibitor (CDKI); G1/S checkpoint; Cell proliferation; Skin disease; Pathogenesis
• 中文摘要	蟹足腫是在人體皮膚上常見的皮膚增生不正常,蟹足腫常因爲皮膚傷害而引起纖維母細胞過度增生,雖然蟹足腫並非惡性皮膚疾病,然而蟹足腫的纖維母細胞可以在裸鼠身上生長,由此推論不受控制的增生可能是因爲由正常細胞週期所逃脫而在於細胞週期中是由週期素依賴性激素抑制者所調控,其中包括有 p21 和 p27。P21 是 p53 下遊的基因,p27 是在於細胞週期推動中伴演週期素/週期素依賴性激素的抑制者。我們研究蟹足腫形成的分子機制,當比較蟹足腫與正常皮膚組織時,我們發現在蟹足腫的皮膚組織中有 p21 以及 p27 的 mRNA 表現,由這結果可以得知控制 G1/S 的控制點的基因在蟹足腫的致病病因扮演重要的角色。
• 英文摘要	Keloid represents one of the most common skin proliferation disorders in humans. The keloid formation usually occurs as a result of hyperproliferation of fibroblasts after skin injury. Even though it is not considered a mailgnant skin disorders. However, the keloid fibroblasts can be successfully implanted and grown in Nude mice. The results suggest an uncontrolled cellular proliferation process which escape the normal cell cycle control. The cells from progressing into S phase which regulated by CdkIs such as: p21 and p27 genes. p21 gene was an downstream regulator of p53 which can also control cell cycle progression. p27 gene was the inhibitor of cyclin/CDK complex which also controlled the cell cycle progression. We were investigated the molecular mechanisms of keloid formation. When compare the keloid with normal control skin tissue, We found the p21(2/6) and p27(2/6) genes mRNA was expression in keloid skin tissue. This result may show the p21 and p27 were involved in controlling G1/S checkpoint play a role in the pathogenesis of keloid.