# 二氫比啶誘發之牙齦纖維母細胞過度增生的機轉中 IL-6/STAT3 訊息傳遞鏈之自動交互作用

# IL-6/STAT3 Autocrine Signaling Pathways in The Cells Derived from Dihydropyridine Induced Gingival Overgrowth


**中文摘要**

團隊近年的研究發現 IL-1β 爲二氫比啶（dihydropyridine）促進牙齦過度增生(dihydropyridine induced gingival overgrowth, DIGO)的主要的促發炎細胞激素。在 DIGO 細胞中，IL-1β 刺激後會增加 IL-6 和睪固酮受器(androgen receptor,AR)之 mRNA 表現。而分別抑制 IL-6 或抑制 AR 皆會降低總膠原蛋白(total collagen)的生成量，並降低結締組織生長因子(Connective tissue growth factor,CTGF)或第一型膠原蛋白(type I collagen)的 mRNA 表現。故 IL-6 與 AR 是影響牙齦增生機轉中的重要因素。本研究欲探討牙齦增生細胞之 IL-6/gp130/STAT3 訊息傳遞鍊與 AR 之關聯。實驗方法將健康的牙齦纖維母細胞與 DIGO 細胞，培養在 charcoal stripped serum 中，分別以不同濃度與不同刺激時間的 IL-6 刺激，進行西方墨點法與雷射共軛焦顯微鏡檢測 STAT3 的磷酸化與 AR 的蛋白質表現與位置。結果顯示健康的牙齦纖維母細胞與 DIGO 細胞 STAT3 磷酸化皆於 15 分鐘內達到高峰，且 DIGO 細胞受 IL-6 刺激產生的 STAT3 磷酸化作用較強。AR 受 IL-6 刺激後的反應在本刺激條件下未產生顯著差異。需進行更進一步的實驗探討 STAT3 與 AR 之間的交互作用。

**英文摘要**

Our previous studies found that IL-1βis the main cytokine which induces Dihydropyridine Induced Gingival Overgrowth (DIGO) in patient with hypertension. Besides, there were more mRNA expressions of IL-6 and androgen receptor(AR) after stimulation of IL-1β in DIGO cells than healthy cells. Therefore, we supposed that IL-6 and AR are main factors of dihydropyridine induced gingival overgrowth. The propose of this study is to find the relationship of IL-6/STAT3 signaling pathway and AR in DIGO cells. Two types of gingival fibroblast, DIGO cells and Healthy cells, were cultured in charcoal stripped serum and treated with different time and dose of IL-6 cytokine. The result of Western blot and Laser confocal microscopy shows that IL-6 induced STAT3 phosphorylation was occurred in 15 minutes and the phosphorylation peak is between 3 and 15 minutes. The STAT3 phosphorylation is more significant in DIGO cells than healthy cells. However, the change of AR is not significant in this study. We have design further experiments to analysis the relationship between AR and IL-6/STAT3 signaling pathway.