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• 計畫英文名稱	Effect of Tumor Necrosis Factor-x on the Electrophysiological Characteristics and Arrhythmogenic Activity of Pulmonary Vein Cardiomyocytes
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• 中文關鍵字	肺靜脈; 腫瘤壞死因子; 電生理
• 英文關鍵字	Pulmonary vein; Tumor necrosis factor; Electrophysiology
• 中文摘要	腫瘤壞死因子乃是一種重要之發炎物質與心血管之致病或心律不整之病理機轉有關。肺靜脈已知是引發心房顫動之重要病灶所在，因此，腫瘤壞死因子或許藉著增加肺靜脈心肌細胞引發心律不整活性而引起心房顫動，本研究之主要目的乃在於探討腫瘤壞死因子對肺靜脈之電生理特性、細胞膜離子電流以及細胞內鈣離子作用。 方法：藉全細胞箝定實驗可以記錄兔肺靜脈心肌細胞之動作電位、離子電流。而細胞內鈣離子乃藉鈣離子螢光偵測加以測量，並比較肺靜脈心肌細胞有無接受腫瘤壞死因子 25ng/ml(6~12 小時)所出現之差異。結果：肺靜脈心肌細胞之對照組與實驗組有相同之自動節律（ 1.9±0.3 Hz 比上 1.8±0.3 Hz），然而，接受腫瘤壞死因子之肺靜脈心肌細胞有較大之動作電位去極化後電位 (5.2±1.6 比上 1.7±0.6 mV)。肺靜脈心肌細胞接受腫瘤壞死因子可以出現較小之 L 型鈣離子流，而有較大之暫時性外向鉀離子流、暫時性離子內流以及鈉鈣交換離子流，再則肺靜脈心肌細胞接受腫瘤壞死因子，有較小之鈣離子流變動，較小之收縮鈣離子。 結論：腫瘤壞死因子可增加肺靜脈心肌引發心律不整活性，這些結果可以解釋發炎合併心房顫動之病理機轉。
• 英文摘要	Background: Tumor necrosis factor-？\ (TNF-alpha), a proinflammatory cytokine, has been implicated in the pathogenesis of cardiovascular diseases and cardiac arrhythmia. Pulmonary veins (PVs) were known to initiate paroxysmal atrial fibrillation. It is known that inflammation plays an important role in the genesis of atrial fibrillation. Therefore, it is possible that TNF-alpha may increase the PV arrhythmogenic activity to induce atrial fibrillation. The purpose of this study is to investigate the effects of

TNF-alpha on PV electrophysiological characteristics, membrane currents and intracellular calcium. Methods: Whole-cell patch clamp were used to investigate the action potentials and ionic currents in isolated rabbit PV single cardiomyocytes with and without (control) incubation of 25 ng/ml TNF-alpha (6~ 12 hours). The intracellular calcium was measured through the indo 1 fluorimetric ratio technique in both groups. Results: There were similar spontaneous beating rates (1.9 .plmin. 0.3 Hz versus 1.8 .plmin. 0.3 Hz) between control (n=30) and TNF-alpha group (n=19). However, PV cardiomyocytes with TNF-alpha had larger amplitudes of delayed afterdepolarization than control cardiomyocytes (5.2 .plmin.1.6 vs. 1.7 .plmin. 0.6 mV, P<0.05). TNF- ? \ group has smaller L-type calcium currents, but has larger transient outward potassium, transient inward, and Na+ - Ca2+ exchanger than control group. TNF-alpha group has smaller intracellular calcium transient, smaller intracellular systolic calcium and longer decay potion of calcium transient (Tau). Conclusions: This study demonstrated that TNF-alpha increases PV arrhythmogenic activity, which may account for the genesis of inflammation related atrial fibrillation.