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• 計畫中文名稱	大豆蛋白水解物對於自發性高血壓大白鼠血壓及血管內皮功能的影響	
• 計畫英文名稱	Effects of Soybean Protein Hydrolysates on Blood Pressure and Endothelium Function in Spontaneously Hypertensive Rats	
• 主管機關	行政院國家科學委員會	• 計畫編號 NSC94-2320-B038-040
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• 中文關鍵字	大豆、蛋白質、血壓	
• 英文關鍵字	Soy, Protein, Blood pressure, NO, ACE, PAI	
• 中文摘要	<p>高血壓為人類最盛行的健康問題，也與心血管疾病、糖尿病等許多慢性疾病間具有相關性，若能在日常飲食中多加注意，應可達到預防和改善的效果。體內調節血壓的一個重要的機制就是腎素-血管收縮素系統 (RAS)，其中血管收縮素轉換酶 (angiotensin converting enzyme, ACE) 可將血管收縮素 I 作用生成血管收縮素 II，長期暴露於高量的血管收縮素 II 會造成血壓升高且可能導致心臟和血管組織的發炎和肥大。本實驗將大豆蛋白以胃蛋白酶水解後進行透析；再將此水解產物添加於利用 L-NAME(N<math>\omega</math>-nitro-L-arginine methyl ester) 誘發高血壓和心腎血管系統病變的大白鼠，以觀察其對血壓和心血管系統的影響。結果發現投予 L-NAME 六週後確實會造成大白鼠血壓上升，而餵食實驗動物大豆蛋白水解物可改善血壓上升的情形。雖然各組間血漿 ACE 活性和 PAI-1 濃度並無顯著差異，但是餵食大豆蛋白水解物的實驗動物心臟和腎臟組織中 ACE 活性和 TNF-<math>\alpha</math> 的濃度都顯著較低；血漿中肌酸酐的濃度也較低。同時我們也發現餵食大豆蛋白水解物的實驗動物週邊組織中脂質過氧化物 MDA 的濃度也較低。在組織病理分析結果中我們也一致的發現餵食大豆蛋白水解物的組別心臟和腎臟組織中發炎的情形也較不嚴重。由本實驗結果我們發現大豆蛋白水解物在體內可改善抑制 NO 所造成的高血壓，另外可能可以藉由抑制組織中 ACE 的活性，而減少組織產生過氧化和發炎肥大的情形，因此有助於避免高血壓的惡化。</p>	
• 英文摘要	<p>Hypertension is a common public problem in many countries and it is related to many chronic diseases. Management of eating habits may be effective in the prevention and retarding the development of hypertension. Renin-angiotensin system (RAS) plays an important role in the regulation of blood pressure in vivo. Angiotensin converting enzyme (ACE) can catalyze angiotensin I into</p>	

angiotensin II and lead to an elevating of blood pressure by arteriole contraction. Long-term exposure to high level of angiotensin II can also lead to endothelium dysfunction and inflammation and hypertrophy of the heart and kidney. We added 1, 3 and 5% of hydrophilic pepsin-digested soy protein hydrolysate (SPH) into diet of L-NAME-induced hypertensive rats. The SPH added did not affect food intake but retarded the development of hypertension and increases plasma NO<sub>x</sub> concentration. Although no significant difference was found in plasma ACE activity, rats fed with SPH had lower heart, lung and kidney ACE activity and TNF- $\alpha$  concentration. Besides, malondialdehyde (MDA) levels in heart, aorta and kidney in rats fed with SPH were also lower than the control group. In pathohistological analysis, we found that the inflammation and hypertrophy of small arteries of heart and kidney in L-NAME treated rats was improved by feeding with SPH. These results suggested that SPH may have retarded L-NAME-induced hypertension and protect the cardiac and renal tissue from oxidative injuries and inflammation by its inhibition effect of tissue ACE activity.