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• 計畫中文名稱	1,3-Beta-D-Glucan 侵入肺部所引發肺迷走 C 纖維感覺神經過度敏感發炎機轉的探討(II)
• 計畫英文名稱	Mechanisms Underlying Hypersensitivity of Pulmonary C-Fiber Afferents Induced by Lung Exposure to 1,3-Beta-D-Glucan (II)
• 主管機關	行政院國家科學委員會
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• 中文關鍵字	呼吸道過度反應症; 葡萄聚醣; 肺 C 纖維感覺神經
• 英文關鍵字	Airway hyperresponsiveness; Glucan; Pulmonary C-fiber afferents
• 中文摘要	吸入 1,3-beta-D-glucan(一種致病黴菌細胞壁的必要成分)會導致氣道過度反應症。然而，形成此過度反應症的機制仍不清楚。肺迷走 C 纖維感覺神經分布於所有呼吸道，並且能偵測呼吸系統的外來入侵物質。刺激此類感覺神經會引發許多呼吸反應，例如：氣道收縮及氣道過度敏感。本研究的目的是探討：肺 C 纖維感覺神經在 1,3-beta-D-glucan 引發的氣道過度敏感中的重要性。我們以麻醉、麻痺並以呼吸氣維持呼吸的天竺鼠爲動物模式，比較在氣管注入 1,3-beta-D-glucan 或它的溶劑之前及之後的 120 分鐘，頸靜脈注射辣椒素(一種 C 纖維專一性刺激物)溶液所引發的氣道收縮反應強度。結果發現，給予 1,3-beta-D-glucan 後，相同劑量的辣椒素注射明顯地引發較強烈的氣道收縮反應；然而，若給予的是 1,3-beta-D-glucan 的溶劑，卻無此增強效果。此增強效果在迷走神經辣椒素包圍法已阻斷 C 纖維傳導後，則大幅減弱。另外，我們的電生理實驗顯示，肺迷走 C 纖維感覺神經的敏感度，在氣管注入 1,3-beta-D-glucan 後，也會大幅提昇。綜合以上實驗結果指出，氣管灌注 1,3-beta-D-glucan 同時將引發呼吸道過度敏感，及肺迷走 C 纖維感覺神經的敏感化。至於，此感覺神經的敏感是否參與在呼吸道過度敏感，將待本計劃後續研究的探討。
• 英文摘要	Airway exposure to 3-beta-D-glucan, an essential component of the cell wall of several pathogenic fungi, is reported to leading to airway hyperresponsiveness. However, the mechanism underlying the pathogenesis of this airway hyperresponsiveness is still unknown. Bronchopulmonary vagal C-fiber afferents innervate all level of the respiratory tract and play an important role in detecting the insults to the lung. Stimulation or sensitization of these C-fiber afferents is known to elicit a number of important reflex responses including bronchoconstriction and airway hyperresponsiveness. The present study was undertaken to evaluate the role of pulmonary C-fiber afferents in the

airway hyperresponsiveness caused by airway exposure to 1,3-beta-D-glucan. Airway constrictive responses to bronchoconstrictor challenge were compared before and 120 min after intratracheal instillation of 1,3-beta-D-glucan or its vehicle in anesthetized paralyzed, and artificially ventilated guinea pigs. 1,3-beta-D-glucan significantly augmented the bronchoconstrictive responses elicited by to right atrial injection of capsaicin, a selective stimulant of C fibers, whereas vehicle of 1,3-beta-D-glucan failed to do so. After perineural capsaicin treatment, Glucan-induced the augmentation of bronchoconstricitve response was largely abolished, suggesting the contribution of vagal C fibers. Furthermore, our electrophysiological data showed that the excitability of pulmonary C-fiber afferents was greatly elevated by intratracheal instillation of 1,3-beta-D-glucan. In summary, these results indicate that airway exposure to 1,3-beta-D-glucan induces airway hyperresponsiveness and the sensitization of pulmonary C-fiber afferents. However, whether sensitization of these afferents is involved in this airway hyperresponsiveness requires further investigation of our ongoing study.