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• 中文關鍵字	抗氧化劑；非膽鹼激性神經系統；一氧化氮；呼吸道；天竺鼠；氧自由基
• 英文關鍵字	Antioxidant；Non-cholinergic nervous system；Nitric oxide (NO)；Respiratory tract；Guinea pig；Oxygen free radical
• 中文摘要	本研究主要探討過氧化物游離基是否會破壞抑制性 NANC 神經系統的保護性角色;以及在非膽鹼性神經之末梢或神經纖維附近,是否存在有內生性可保護 NO 自由基的抗氧化系統。我們發現在天竺鼠的氣管,過氧化物(Superoxide anion)的產生者如 Pyrogallol 和 Duroquinone 以劑量相關的方式抑制 S-nitroso-N-acteyl-penicillinamine(SNAP)所引起的平滑肌鬆弛反應,但對於直接電刺激 NANC 神經,所引起的平滑肌鬆弛反應則沒有抑制作用。NO 的清除者如 Carboxy-PTIO 也以劑量相關的方式抑制 SNAP 所引起的平滑肌鬆弛反應;同樣地,對於直接電刺激 NANC 神經,所引起的平滑肌鬆弛反應也沒有抑制作用。抗氧化劑 Cu/Zn SOD 和 Ascorbate 皆可減少 Pyrogallol,Duroquinone 對於 SNAP 的抑制反應;同樣地,Cu/Zn SOD 和 Ascorbate 也減少 carboxy-PTIO 對於 SNAP 的抑制反應。而當天竺鼠氣管環標本預先給予 Cu/Zn SOD 的抑制劑 Diethyldithiocarbamate(DETC,3mM)前處理 45 分鐘後,可發現 Pyrogallol 和 Duroquinoneru 皆可部分抑制電刺激所引發的氣管鬆弛反應,而這個作用可被 Cu/Zn SOD 和 Ascorbate 所抑制。由以上之結果得知 Pyrogallol 和 Duroquinone 並不抑制電刺激 NANC 神經所引起的平滑肌鬆弛反應,但是當 Cu/Zn SOD 被 DETC 抑制後,Pyrogallol 和 Duroquinone 即可部分抑制電刺激所引發的氣管鬆弛反應,這結果暗示在氣管的 NANC 神經末梢,存在有高濃度的抗氧化物質可以保護 NANC 的神經傳遞物質 NO,使其免於受到 Superoxide anion 的攻擊。同時亦可提供控制呼吸道鬆弛反應的一個新方向。
• 英文摘要	In this study, we will investigate the hypothesis that NO breakdown may be increased by oxygen free radical, thus leading to the destruction of the protective role of inhibitory NANA nerve. Furthermore, we will also study whether the presence of endogeneous

antioxidant system in the vicinity of the neuroeffector junction may protect the endogeneous transmitter from attack by oxygen free radical. The superoxide anion generator, pyrogallol and duroquinone, significantly inhibited the tracheal relaxation to S-nitroso-N-acetyl-penicillinamine (SNAP) but not nitrergic relaxations to NANA nerve stimulation. SOD and ascorbate can reversed the inhibition of pyrogallol and duroquinone on SNAP-induced tracheal relaxation. The NO scavenger, carboxy-PTIO also significantly inhibited the tracheal relaxation to SNAP but not nitrergic relaxations to NANA nerve stimulation. Similarity, SOD and ascorbate can reversed the inhibition of carboxy-PTIO on SNAP-induced tracheal relaxation. Following inhibition of endogeneous Cu/Zn SOD activity with diethyldithiocarbamate (DETC, 3mM), pyrogallol and duroquinone, also inhibited the tracheal relaxation to NANA nerve stimulation, and this effect was also reversed by SOD and ascorbate. These results suggested that the presence of endogeneous antioxidant system in the vicinity of the neuroeffector junction may protect the endogeneous transmitter from attack by oxygen free radical. These studies also provided evidence for the modulatory role of exogeneous and endogeneous antioxidant system in inhibitory NANC nerve and may provide new ways of controlling the relaxation of tracheal smooth muscle.