大豆油與 **b-**胡蘿蔔素對大白鼠初代肝細胞脂質過氧化及抗氧化酵素活性的影響

Effects of soybean oil and beta-carotene on lipid peroxidation and antioxidant enzymes activities in primary rat hepatocytes

中文摘要

大豆油是膳食中，多元不飽和脂肪酸的主要來源。研究指出，增加多元不飽和脂肪酸的攝取，會促進體內脂質過氧化的發生。而 -胡蘿蔔素是飲食中，含量較高的一種類胡蘿蔔素，具有清除單態氧，及延緩脂質過氧化的抗氧化能力。因此，利用管灌的方式，每日投予大白鼠 400 L 大豆油及 40 mg -胡蘿蔔素，餵養 4 週後，以大白鼠初代肝細胞爲實驗模式，添加次亞麻油酸，使細胞培養液中次亞麻油酸最終濃度爲 0.01，0.1，0.5 或 1 mM，提高細胞脂質過氧化的發生，以觀察大豆油及 -胡蘿蔔素，對脂質過氧化的影響。實驗結果發現，以含有 0.5 和 1 mM 次亞麻油酸的培養液培養，顯著增加細胞內丙二醛濃度，降低抗氧化酵素中，超氧化物歧化酉每 和麩胱甘月太 過氧化酉每 的活性。補充大豆油，降低麩胱甘月太 過氧化酉每 及觸酉每 的活性，且增加細胞中亞麻油酸及次亞麻油酸的比例。補充 -胡蘿蔔素，增加細胞內 -胡蘿蔔素的含量，但以次亞麻油酸培養 6 小時後，則降低至無法測得。 -胡蘿蔔素減緩空白組和以 0.01 mM 次亞麻油酸培養的細胞，丙二醛濃度的增加，但卻會加速以 0.1 mM 次亞麻油酸培養的細胞，丙二醛的產生。 -胡蘿蔔素的補充，亦具有降低超氧化物歧化酉每 活性的影響。推論，因大豆油的補充，增加細胞中不飽和脂肪酸的比例，提高細胞對於自由基攻擊的感受性和前列腺素合成的反應，導致麩胱甘月太 過氧化酉每 及觸酉每 活性的降低。 -胡蘿蔔素在氧化壓力下，具有促氧化的影響，因此降低超氧化物歧化酉每 的活性，加速以 0.1 mM 次亞麻油酸培養的細胞，丙二醛的產生。根據實驗結果指出，在本研究實驗模式下，大豆油的補充，加速脂質過氧化的發生，降低麩胱甘月太 過氧化酉每 的活性。 -胡蘿蔔素因其抗氧化力不同的表現，對於脂質過氧化有延緩及促進的影響，且降低超氧化物歧化酉每活性。

英文摘要

Soybean oil is the main source of dietary polyunsaturated fatty acids. Several studies have shown that high intake of polyunsaturated fatty acids promotes lipid peroxidation. -carotene (BC) is one of the prevalent carotenoids in diets and also a scavenger of singlet oxygen. Indices of lipid peroxidation are lowed by BC supplements. In order to investigate the effects of soybean oil and BC on lipid peroxidation, rats were supplemented 400 L soybean oil and 40 mg BC daily by gavage for 4 weeks. Then, hepatocytes were isolated by two-step collagenase perfusion and incubated in medium which contained 0.01, 0.1, 0.5 or 1 mM -linolenic acid (ALA) to induce lipid peroxidation. Results indicate that medium contained 0.5 or 1 mM ALA increased cellular malondialdehyde (MDA) concentration and decreased superoxide dismutase (SOD) and glutathione peroxidase (GSHPx) activities significantly. Soybean oil supplementation decreased GSHPx and catalase activities and raised the ratio of linoleic acid and ALA within the cell. BC supplementation enhanced cellular BC content but 6 hour-ALA incubation reduced it to an undetectable level. BC declined SOD activity and had the ability to delay MDA augmentation in cells which were incubated in medium without or with 0.01 mM ALA. We suppose that soybean oil supplementation decreased GSHPx and catalase activities by higher polyunsaturated fatty acids ratio within the cell which might increase susceptible to free radicals attack and promote prostagladin synthesis. Under oxidative stress, BC had prooxidant property to lower SOD activity, and speed up MDA accumulation in cells incubated in 0.1 mM ALA medium. We conclude that in this experimental model, soybean oil supplementation promotes lipid peroxidation and decreases GSHPx activity by increasing cell susceptible o free radicals. Effect of -carotene on lipid peroxidation and SOD activity is dependent on it’s antioxidative ability.