

添加鐵與不同飽合度之油脂對低密度脂蛋白氧化修飾的影響

葉羨敏

以往研究認為，食用愈飽和的油脂較易引起動脈粥狀硬化，食用較不飽和油脂則可降低血中的脂質並避免此病的發生。但不飽和油脂較易氧化，而低密度脂蛋白(LDL)的氧化修飾是引發動脈粥狀硬化的成因之一，則較不飽和的油脂反而有可能容易導致動脈粥狀硬化。為了避免貧血，醫師或營養師常建議補充鐵劑，但過多的鐵是否會因促進 LDL 的氧化而變得不利，均值得探討。本研究重點即擬採體內(In vivo)和體外(In vitro)的方法，來探討攝取較不飽和的油，是否因其 LDL 所含的脂肪酸較不飽和，而進一步較易被氧化？以及當攝取較多的鐵質時，縱使仍未造成血中鐵濃度升高，是否已足以促進 LDL 的氧化？本研究以倉鼠為實驗動物，攝取添加 15%棕櫚油、黃豆沙拉油和魚油等不同飽和度食用油，並同時給予 1 倍 AIN(American Institute of Nutrition)或 4 倍 AIN 的鐵建議量，作為低和高鐵攝取量。以探討這些因子是否會影響到血清 LDL 的氧化。飼養 8 週結束後，取血樣，除直接測定 LDL 過氧化脂質外，並以體外添加鐵方式加速 LDL 的氧化，來比較攝取不同飼料之動物，其 LDL 氧化敏感度是否有差異。測定方法乃以共軛雙鍵產物的產生量作為血漿 LDL 氧化程度的指標，實驗結果顯示魚油組較其他實驗組有較高之共軛雙鍵的形成，而攝取高鐵高黃豆油組的起始所含共軛雙鍵產物也顯著高於($P > 0.05$)低鐵黃豆油組、棕櫚油組與控制組。此外，以高效液相層析儀配合電化學偵測分析動物體內 LDL 脂質過氧化產物，發現：高魚油組 LDL 所含的脂質氫過氧化物較多，高鐵高魚油組之脂質氫過氧化物又顯著高於其他各組($P > 0.05$)。但倉鼠血清鐵、輸鐵蛋白總結合力和輸鐵蛋白飽和度則於各實驗組間無顯著差異($P > 0.05$)。由本研究結果可知，大量攝取高鐵及富含高不飽和度油脂飲食，雖然並無反映在血清鐵與輸鐵蛋白總合力上，但在體內及體外實驗中，卻已可能促進 LDL 脂質過氧化，在動脈粥狀硬化的病理發生上，高鐵配合高不飽和度油脂的攝食方式，可能具有潛在的危險性。

來源:

<http://metadata.ncl.edu.tw/blstkmc/blstkmc?002B336EC1D3000200000000000103200000003E00000000^NO#tudorkmtop>

<http://elib.lib.nttu.edu.tw/theses/brlist.asp?br=Sname&ID=%B8%AD%B8r%B1%D3>

<http://scholar.fju.edu.tw/paper/002414.html>



Effects of Adding Iron and Various Saturation Edible Oils on Oxidation of Low Density Lipoprotein

Hsien-min Yeh

It has been demonstrated that feeding with saturated fats may elevate plasma low density lipoprotein (LDL) cholesterol and induce atherosclerosis. Unsaturated fat feeding exhibits an opposite effect. Recent studies have indicated that oxidized low density lipoprotein (OX-LDL) plays a pivotal role in the pathogenesis of atherosclerosis. Since unsaturated fats are more susceptible to oxidation, it is reasonable to suspect that feeding with unsaturated fats may increase LDL oxidation and atherosclerosis. Iron supplementation is commonly recommended by medical doctors and nutritionists to treat anemia. However, the possibility that LDL oxidation may be accelerated by iron can not be overlooked. This thesis intends to elucidate if unsaturated fats feeding combined with high iron uptake may further induce LDL oxidation. Hamsters were chosen as the animal model. Animals were divided into seven groups and each fed with a control diet or an experimental diet containing 15% palm oil, soybean oil, or fish oil for 8 weeks. To each experimental diet group, either a 100% or 400% AIN iron as iron (III) citrate was added. LDL oxidation in vitro was induced by Fe(3+) with the formation of conjugated dienes as the marker. Results showed that LDL from animals fed with fish oil exhibited high conjugated diene formation. Basal conjugated diene level was also higher in LDL from the high iron soybean oil group than, that of low iron soybean group ($p < 0.05$). Lipid hydroperoxide contents in LDL as determined by HPLC-ECD were higher in fish oil groups than in other groups. Lipid hydroperoxide level in LDL from animals fed with a high iron fish oil diet was also significantly higher ($p < 0.05$). There was no significant difference in serum iron concentration, total iron binding capacity, or transferrin saturation among the animal groups. This study concludes that high iron feeding (400% AIN Fe(3+)) to hamster may not affect serum iron concentration or total iron binding capacity. However, LDL oxidation in vitro and ex vivo is increased significantly. On the basis of LDL oxidation hypothesis, high iron uptake may impose latent risk on the pathogenesis of atherosclerosis.

Source:

<http://metadata.ncl.edu.tw/blstkmc/blstkm?002B336EC1D300020000000000010320000003E00000000^NO#tudorkmtop>

<http://elib.lib.nttu.edu.tw/theses/brlist.asp?br=Sname&ID=%B8%AD%B8%B1%D3>

<http://scholar.fju.edu.tw/paper/002414.html>

