High fat diets do not increase CCl_4-induced oxidative stress in the rat

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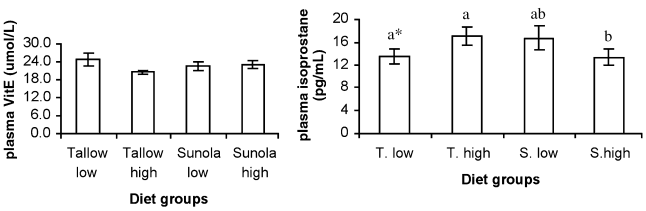
High fat diets have been associated with increased obesity and other chronic lifestyle diseases such as atherosclerosis and type II diabetes. High fat diets may promote atherogenesis by subjecting vascular endothelial cells to oxidative stress (1). Due to discoveries of potential relationships between oxidative stress, activation of oxidative stress-sensitive transcription factors and the etiology of acute and chronic diseases, the issue of the type of dietary fat and the amount of dietary fat consumed needs to be revisited (1). This study was conducted to examine the effect of high fat diets as saturated or monounsaturated fatty acids on oxidative stress.

Four groups of rats (n = 9) were fed either a high fat (200 g/Kg diet) or a low fat (50 g/Kg diet) diet based on Tallow (saturated) or Sunola (monounsaturated). The rats were fed the diet for four weeks, then gavaged with CCl_4 to induce oxidative stress. The rats were euthanised four hours later and plasma and liver collected for analysis.

Oxidative stress, as indicated by the plasma isoprostane concentrations (8-iso-$PGF_{2\alpha}$), were not affected by the increased fat in the diet. There was a significant decrease in plasma isoprostane values between the high fat Sunola and both the low fat and high fat Tallow diet groups ($p < 0.05$). However, isoprostane concentrations were unaltered by the amount and type of fat in the liver tissue. The increase in fat did not affect either plasma vitamin E, or liver vitamin E concentration. In combination these two parameters indicate that there was no increase in oxidative stress caused by the increase in the amount of fat in the diet regardless of the type of fat in the diet.



*The values without common superscript are significantly different ($p<0.05$)

In conclusion, there is no increase in oxidative stress due to an increase in amount of fat in the diet. It appears that when an increase in oxidative stress occurs it is probably due to the type of fat in the diet, not necessarily the amount of fat in the diet.

Reference

1. Hennig B, Toborek M, and McClain CJ. High energy diets, fatty acids and endothelial cell function: implications for atherosclerosis. J Am College Nutr, 2001; 20: 97–105.

Supported by a joint grant from Meadowlea Foods and GRDC
Key words: high fat diets, oxidative stress, F_2-isoprostane