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Lack of correlation between plasma and prostate tissue alpha-linolenic acid levels

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Background - Several epidemiological studies have reported a positive association between plasma alpha-linolenic acid (ALA) levels and the incidence of prostate cancer; however other studies have not supported this association, as recently reviewed.[1]
Objective - The aim of this study was to determine if there was a correlation between the plasma and prostate tissue levels of ALA and/or other n-3 polyunsaturated fatty acids (PUFA).
Design - Plasma and prostate tissue were collected from patients undergoing prostate surgery, and prior to surgery completed a brief dietary questionnaire on the intake of dietary n-3 PUFA. Twenty-eight patients participated in the study, of which 20 were diagnosed with benign prostatic hyperplasia (BPH) and 8 with prostate cancer (PC).
Outcome - The main lipids in the prostate tissue were phospholipids (PL) and sterols. The results showed that there was no significant correlation between plasma PL ALA and prostate tissue PL ALA concentrations (or proportions). There were, however, positive correlations between the proportions (not concentrations) of plasma and prostate tissue for EPA, DHA and total n-3 PUFA in the PL fraction. There was no significant difference in plasma and prostate tissue ALA and n-3 PUFA levels between the BPH patients and the PC patients for either the PL and triacylglycerol (TAG) fractions. Plasma ALA concentrations were significantly higher than the prostate tissue ALA levels for each subject.
Conclusions - These pilot data do not show a significant association between plasma and prostate ALA in humans.

1. Attar-Bashi NM, Frauman A and Sinclair AJ. (2004) Alpha-Linolenic Acid and the Risk of Prostate Cancer, What is the Evidence? Journal of Urology, 171:1402-7.

Docosa-hexaenoic acid (DHA) accumulation is regulated by the polyunsaturated fat content of the diet: Is it synthesis or is it incorporation?

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Background - Tissue levels of docosahexaenoic acid (DHA, 22:6n-3) in animals and humans are minimally influenced by increasing the level of its precursor alpha linolenic acid (ALA, 18:3n-3) in the diet. We have tested the hypothesis that this could be due either to competitive inhibition of a key step in the pathway, the conversion of dietary ALA to long chain polyunsaturated fatty acids (LCPUFA) by linoleic acid (LA, 18:2n-6) since both fatty acids (ALA, LA) are substrates for the Δ^6-desaturase or is due to LA inhibiting DHA incorporation.
Methods - We tested weaning rats fed a spectrum of 54 separate diets for three weeks. The diets varied in fat content (11.8, 22.2 and 39.4 percent of energy, en%), in the levels of LA (0.07 - 17.1 en%), ALA (0.02 - 12.1 en%) and in the LA:ALA ratio (0.5:1 to 10:1).
Results - The concentrations of DHA in plasma phospholipids of some dietary groups reached 9% of total fatty acids but the peak of DHA accumulation was seen within a narrow range of 1-3 en% ALA and 1-2 en% LA. Beyond 3 en% of either ALA or LA, DHA levels are uniformly low. On the other hand, plasma DHA levels were inversely correlated with plasma LA (r^2=0.6) indicating that high LA intakes may inhibit incorporation of DHA. This may explain the apparent curvilinear effect of dietary ALA on synthesis of DHA. Past stable isotope experiments may not taken this into account.
Conclusions - The apparent conversion of ALA to DHA is dependent on both an adequate level of ALA and a low level of LA in the diet.