

# **Circulating aP2 and adipocyte aP2 gene expression as predictors of metabolic response during a weight loss program in obese adults**

**Project: 410**

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## **Background**

aP2, traditionally considered to be an intracellular cytosolic protein, is suggested to be a plasma biomarker associated with the metabolic syndrome. However, several major questions about the metabolic role of aP2 remain unanswered. The aim of this study was to investigate associations between circulating aP2, adipocyte aP2 mRNA expression, body weight and metabolic risk factors in obese adults during weight loss.

## **Methods**

In obese adults participating in two different weight-loss programs, we measured changes in anthropometric and metabolic parameters, subclinical inflammation, circulating aP2, and adipocyte aP2 mRNA expression, and examined relationships among these variables.

## **Results**

Circulating aP2 is markedly increased in obese adults. Baseline circulating aP2 concentrations positively correlated with BMI ( $p=0.006$ ). However, no other correlation was found between circulating aP2, weight and metabolic parameters at baseline. Circulating aP2 concentrations increased dramatically (by 81%) with weight loss, from a median aP2 concentration of 38.1 (18.8-106.1) ng/dl at baseline to 69.1 (15.4-145.4) ng/dl after 3-month weight loss ( $p<0.001$ ). Moreover, a negative correlation between  $\Delta$  circulating aP2 and  $\Delta$  weight was observed ( $r_s=-0.506$ ,  $p=0.003$ ). Thus, the higher the weight reduction was, the higher the increase in circulating aP2 during the 3-month time period. Also adipocyte mRNA aP2 expression increased with weight loss, from 28.8 (3.5-68.5) pg/ $\mu$ g cDNA at baseline to 100.0 (52.0-156.4) pg/ $\mu$ g cDNA after 3 months ( $p<0.001$ ).

## **Conclusion**

During short-term weight loss there is an increase in both circulating aP2 concentrations and aP2 expression in adipocytes. These changes suggest an intracellular, rather than an extracellular, role of aP2 in fatty acid metabolism. They likely are a response to the marked increase in lipolytic activity in adipose tissue during negative energy balance.