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Eucaloric High Fat Diet Does Not induce insulin Resistance But May Stimulate Fat Oxidation in Normal Weight Women

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Obesity and metabolic syndrome are associated with defects in the hypothalamic pituitary ovarian (HPO) axis termed the reprometabolic syndrome. We have previously shown that lipid infusion induces insulin resistance and the reprometabolic syndrome of obesity in normal weight women. We hypothesized that the induced insulin resistance is the underlying cause of the attenuation of the hypothalamic pituitary ovarian (HPO) axis. 16 women of normal BMI (18-24.9g/m2), mean age 29.7 ± 6.4, were recruited for a study including a 30-day, prescribed, eucaloric, high fat (48% of calories from fat) dietary intervention. Diet was adjusted to ensure that participants remained weight stable throughout the study. Insulin sensitivity was measured by 2 stage euglycemic hyperinsulinemic clamp (8 and 40 mU/m^2*min) pre and post diet. Final stage 2 glucoses were not different pre vs post diet (mg/dL, 86±7 vs 89±6, p=0.08). Muscle insulin sensitivity (glucose infusion rate, mg/kg*min) was different pre versus post diet (10.9±3.3 vs 10.6±3.5, p=0.013) after exclusion of one participant for multiple protocol deviations. Adipose insulin sensitivity as measured by fatty acid suppression during the low insulin infusion stage of the clamp was also not different pre versus post diet [final fatty acid level in μEg/L; median 115 (IQR 84, 224) vs 158 (IQR 67,274; p>0.99). However, the final high insulin stage 2 fatty acid suppression trended towards greater suppression after high fat diet [median 17.8 (IQR 9.8,33.8), vs 11.7 (IQR 7.7, 18.7) p=0.06]. This may reflect higher fatty acid oxidation on the high fat diet.

We have previously reported that this diet, like the lipid infusion, did attenuate the HPO axis (reduced baseline early follicular LH and FSH and reduced LH response to GnRH). In contrast to lipid infusion, however, this dietary intervention did not induce the same degree of insulin resistance, possibly due to the emphasis on neutral energy balance, suggesting that the suppression of the HPO axis by high fat diet may be partially independent of insulin sensitivity.

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