Association between obesity and abnormal postprandial hemodynamics in adolescents

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Introduction: Early cardiovascular risk often remains undetected in the young as conventional means of risk stratification commonly rely on characterizing resting physiology. Using a novel dynamic MRI protocol, we hypothesized that ingestion of a high-carbohydrate, high-fat meal can unmask latent hemodynamic dyregulation in order to identify a pro-atherosclerotic state.

Methods: Eighty healthy teenagers (34 females; median age 16, IQR: 14 to 18 years), 34 of which were either overweight (BMI>25) or obese (BMI>30; n=19) underwent the following protocol: After 12 hours of fasting, resting blood pressure (BP) was obtained and, using rapid MRI sequences, blood flow (BF) was measured in segments of the aorta (ascending and supra-iliac) as well as the renal, celiac and superior mesenteric arteries (CA and SMA). Following a high calorie meal (1,600 kcal), these measurements were repeated every 5 to 10 minutes for 1 hour. Interactions between hemodynamic measures and time, BMI Z score for age (zBFA) and sex were assessed in a multilevel regression model.

Results: In the fasting state, there was a significant association between zBFA and systolic BP (2mmHg per zBFA increment, p<0.05), iliac and renal BF (0.09 and 0.08 L/min per zBFA increment, p<0.005 each), and an inverse correlation of cardiac index (CI) with zBFA (0.18 L/min/m2 decrease per zBFA increment, p<0.001). By contrast, fasting perfusion of the SMA, the CA was not correlated with body weight. Following meal ingestion, there was a significant increase in CI, renal, celiac, mesenteric and iliac BF. The increase in BF to the SMA was significantly lower in overweight subjects than in the normal weight (0.13 L/min decrease per zBFA increment after 1 hour, p<0.05). By contrast, the remaining changes in BF were comparable across all weight groups.

Conclusions: For the first time we could demonstrate that postprandial mesenteric perfusion is impaired in overweight adolescents. Our findings suggest a mismatch between the postprandial metabolic demand of the small gut and the cardiovascular response in this population. Further research is necessary to better understand how this dysregulation influences atherosclerotic risk in the young.