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Commentary

Is a Biomarker Microvascular Model for Dilation?

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Is a biomarker microvascular model for dilation?

Does low-fat diet for weight loss make the impaired microvascular dilation improved through switching to nitric oxide (NO)-dependent in obese adults? The answer is yes. Shane Phillips research group has tested this hypothesis through their most recent publication [1]. While systemic NO doesn't change, localized NO was increased after a six-week low-fat diet for weight loss (~25% calorie deficit) but not for weight maintenance to obsess subjects with body mass index (BMI most subjects in 30-35, and only a few in 35-40 kg/m2) through increasing flow-induced dilation of arterioles. These in vitro microvascular model data are consistent with data with clinical increase of flow-mediated dilation and decrease of BMI in post- compared to pre-low-fat diet for weight loss [1, 2]. Interestingly, indomethacin, the cyclooxygenase enzyme inhibitor, reduced flow-induced dilation of arterioles in pre- but not in post-low-fat diet for both weight loss and weight maintenance, suggesting that cyclooxygenase-derived metabolites, inflammatory factors, involves dilation in these obese subjects before a six-week low-fat diet and this involvement is disappeared in post-low-fat diet. Hydrogen peroxide (H2O2) involved partial flow-induced dilation of arterioles through Peg-catalase (the H2O2 scavenger)-reduced dilation, but these reduced dilations have no changes in post- compared to pre-low-fat diet for weight loss in this study (unpublished data). In our early study, a group of subjects in a binge drinking study were recruited with BMI most in 20 to 24 kg/m2 and only a few subjects (three out of total fourteen subjects) more than 25 but less than 30 kg/m2 [3], microvascular dilation of arterioles was only NO-H2O2-dependent while L-NAME, inhibitor of endothelial nitric oxide synthase (eNOS), and together with Peg-catalase, completely blocked this dilation in control (abstainer) group. In binge drinking group, this dilation was reduced through losing NO-dependent and rest of dilation was H2O2-dependent. MicroRNA-21 inhibitor restored this reduced dilation and made the dilation completely NO-dependent but not H2O2-dependent at all. In our earlier morbid obese subject study with BMI only some in 35-40 and most subjects more than 40 kg/m2 [4], as a control, almost 100% baseline dilation of arterioles of subcutaneous adipose tissues were NO-, H2O2-, and cyclooxygenase-derived metabolites-dependent. However, only 40% baseline dilation of arterioles was left in visceral adipose tissues compared to that of subcutaneous adipose tissues. This left dilation was reduced but not significantly by L-NAME and indomethacin, respectively, but not by Peg-catalase. Furthermore, L-NAME and indomethacin, or L-NAME and indomethacin and Peg-catalase significantly blocked more but not completely this left microvascular dilation of visceral fats (unpublished data). Are any other dilator components in nature contributing to this unblocked and left dilation? What are these dilator factors for maintaining the left dilation of arterioles in visceral fats of morbid obese subjects?

Nitric oxide is endothelium-synthesized and secreted and most potent physiological dilator. During and after exercise, microvascular NO-dependent dilation is compensated by H2O2, a by-pass metabolic product during energy supplies. In in vitro microvascular model, this conversion of NO to H2O2 has been mimicked by a transient increase in intraluminal pressure [5]. Using this extended model, we recently found that tetrahydrobiopterin (BH4), a naturally occurring essential cofactor of eNOS, restored the impaired dilation of arterioles in binge drinking subjects during and after increase of intraluminal pressure, indicating deficiency of endogenous BH4 results in uncoupling of eNOS that makes unavailable for production of nitric oxide in binge drinking subjects (Hwang, C-L, Bian J-T, et al, submitted to Microcirculation).

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Conflicts of Interest
The author declares no conflict of interest.

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